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THE GENETIC FACTOR IN OTOSCLEROSIS

CHARLES B. DAVENPORT, PH.D.

BESS LLOYD MILLES, PH.D.

AND

LILLIAN B. FRINK, M.A.

COLD SPRING HARBOR, N. Y.

PART I. PROBLEM, METHODS OF STUDY AND RESULTS

A. STATEMENT OF PROBLEM

1. *Definition of Otosclerosis.*—Otosclerosis may, for the purpose of this study, be defined as a process of change in the bony structure of the otic capsule, located in the petrous portion of the temporal bone (labyrinthine capsule). The changes usually involve, first, an ossification of the annular membrane connecting the margin of the base of the stapes and the margin of the fenestra ovalis (which is closed by the base of the stapes), so that the stapes is firmly ankylosed to the margin of the fenestra ovalis. They lead, ordinarily, to a progressive hardness of hearing.

In recent years the bony changes in otosclerosis have been found to be even more widespread in the petrosal bone than was formerly recognized. Large hollow spaces arise in the bone, which has pathologically enlarged blood vessels and a rather dense network of connective tissue between the bone and the vascular walls (Drury [1]). The pathologic process consists in absorption of old bone, followed by the deposition of new-formed spongy bone in its place. The haversian canals become enlarged, their walls being absorbed by osteoclasts. Then new blood vessels are formed, osteoblasts appear and new bone is laid down. Smaller or larger exostoses are formed. The pathologic process shows no signs of inflammatory changes (Drury).

Functionally, otosclerosis shows itself by a loss of perfect, sharp hearing, which is progressive; it starts usually in one ear but usually eventually involves both. The deafness is accompanied, especially in the earlier years, by tinnitus, usually with paracusis willisiana (improved hearing in the presence of a background of loud noise).[2] The lower vibrations are most apt to be cut out from sensation. The Rinne test gives negative results. In Weber's test the tone is heard better in the

From the Department of Genetics, Carnegie Institution of Washington.

1. Drury, D. W.: Otosclerosis, Ann. Otol., Rhin. & Laryng. **35**:651, 1926.

2. For the sake of brevity, paracusis willisiana will be referred to in this paper as "paracusis."

affected ear, indicating a defect in the conducting apparatus, not in the endings of the auditory nerve. The Schwabach test shows bone conduction normal or increased.

Otosclerotic changes occur in from about 0.1 to 0.25 per cent of the adult white population.[3] The proportion is greater in females than in males in the ratio of 66:34.[4]

3. The question, What proportion of the population carries the factors that constitute the endogenous cause of otosclerosis? is not easy to answer. First of all, a large proportion of such potential cases have not revealed themselves, because the patients have not reached the age of onset, from 15 to 30 years. Second, otosclerosis is not easily defined so as to separate it from other causes that are responsible for hardness of hearing, such as senile deafness, progressive labyrinthine deafness or even hardness of hearing following otitis media. Third, of defects of hearing there are all grades. At what point is a person hard of hearing?

If, in a search for a rough approximation of the probable number we go to the United States census returns of 1900 on the deaf, as revised and reported by Alexander Graham Bell in 1909, we find that 89,287 were enumerated as "deaf." Of these 84,361 were deaf white persons of continental United States in a population of 66,809,196 white persons. This represents about 1.3 deaf persons per thousand of the white population.

But this ratio is not to be regarded as very important. First of all, enumerators were instructed to count as "deaf" those who "were unable to understand loudly shouted conversation." A critical examination of the returns and a reenumeration by correspondence reveals the fact that of these 89,287 "deaf," 37,426 were totally deaf and 51,861 could hear loudly shouted conversation. The latter were classified in Bell's analysis under the head of "partially deaf," although, added Bell (p. 67), "the cases reported constitute only a small fraction of the partially deaf of the country." Again, certain considerations led Bell to say (p. 67): "It is therefore probable that the majority of the partially deaf persons returned were laboring under serious defects of hearing, and were not simply 'hard of hearing.'" The census returns, accordingly, throw little light on the number of deaf and hard of hearing in the population. The total number must be over 1.3 per thousand, and may be of the order of 10 per thousand.

This rough guess does not give any idea of the proportion of the population that is genetically of the otosclerotic constitution, for (as already stated) this constitution does not usually reveal itself until after puberty. Of the "partially deaf" of the census returns Bell found (p. 79) in 46,111 who stated the "age of onset" that the age was under 15 years in 12,593 and 15 or over in 33,518. There were, indeed, 17,763 of the 46,111 persons in whom the "age of onset" was between 15 and 40. This is the range of ages in which the "onset" of otosclerosis commonly occurs. It includes about 40 per cent of the "partially deaf." Assuming half of those becoming "partially deaf" between 15 and 40 years to have otosclerosis, then roughly 20 per cent of the enumerated "partially deaf" are otosclerotic. Also let us assume that the "small fraction" of the reported deaf is one seventh. Then about 0.78 per cent of the population has an unrecorded hardness of hearing. If 20 per cent of these persons also have otosclerosis, we would have

20 per cent of 0.13 per cent = 0.03 per cent
20 per cent of 0.78 per cent = 0.15 per cent

Total = 0.18 per cent

(*Continued on page 137*)

2. *Ontogeny of Otosclerosis.*—The newer studies in genetics are demonstrating that the adult "characters" can usually be traced back in development to very early embryonic stages. An understanding of otosclerosis is aided by a knowledge of the early ontogeny of the otic capsule.

Our knowledge of the chondrification and ossification of the otic capsule in human fetuses depends largely on the studies of Streeter [5] and Bast.[6]

As for the cartilaginous capsule of the ear, Streeter,[7] said:

> Throughout the entire period of growth . . . it was found that the margins of the cartilaginous cavities undergo a process of continual transformation. They exhibit a state of unstable equilibrium in respect to the opposing tendencies toward a deposit of new cartilage on the one hand and toward the excavation of the old, on the other.

As for the later stages, Bast [8] found:

> The process of ossification of the cartilaginous capsule begins by (*a*) enlargement of the cartilage lacunae, (*b*) modified staining reaction of the cartilage matrix, (*c*) entrance of connective tissue cells into the enlarged lacunae which replace the cartilage cells, (*d*) entrance of vascular osteogenetic buds which excavate large channels in this changed cartilage. This excavation is not complete, thus leaving islands of cartilage. Primary endochondral (*e*) bone is formed in the excavated parts; (*f*) the connective tissue cells which wandered into the lacunae of the cartilage islands change into bone cells, surrounding themselves with bone. The cartilage matrix of these islands is calcified, thus producing a peculiar type of bone which we have designated intrachondral bone.

Now otosclerosis is associated with an abnormal bone formation, or rather with reorganization of bone. Of this there may be all degrees. Thus Guild [9] found abortive otosclerotic changes in the otic capsule of a man of 68 years, whose hearing was regarded as normal. In some

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(*Footnote 3, continued from page 136*)

On this basis, about 0.2 per cent of the white population is otosclerotic.

Nager (1928) found otosclerosis in 20 per cent of all of his patients with disease of the ear. One fifth of the deaf persons reported in the census would be 0.03 per cent of the population, but we have given reasons why the census figures should be increased many fold. The foregoing figures are offered as a sort of hypothesis to be tested further.

4. Nager, cited in Duel, A. B.: Otosclerosis, New York, Paul B. Hoeber, Inc., 1929, p. 489; see also table 2.

5. Streeter, G. L.: The Histogenesis and Growth of the Otic Capsule and Its Contained Periotic Tissue-Spaces in the Human Embryo, Contrib. Embryol. (no. 20) **7**:1, 1918.

6. Bast, T. H.: Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. (no. 121) **21**:53, 1930.

7. Streeter (footnote 5, p. 48).

8. Bast (footnote 6, p. 81).

9. Guild, S. K.: Early Stages of Otosclerosis, Arch. Otolaryng. **12**:471 (Oct.) 1930.

cases the degree of impairment is slight throughout life; in still other cases otosclerotic changes are extensive and passed through rapidly. Guild [10] concluded that otosclerotic changes may begin as early as the end of the first year of postnatal life. The exact location of the first appearance of the area near the anterior margin of the oval window is not the same in all cases. An area of otosclerosis may arise near the anterior margin of the oval window, grow to considerable size and become quiescent without the formation of any calcified connections with the base of the stapes. Ankylosis must be well started before a clinical diagnosis of otosclerosis is possible by the present method of examination.

Abnormal growth of other parts of the temporal bone is not uncommonly associated with otosclerosis. Numerous exostoses have repeatedly been found at autopsy in other parts of the petrosal bone than just around the stapes.[11] For example, synostosis occurs between the mastoid process and occipital bone,[12] and thickening is found around the semicircular canals, over nearly all of the petrous bones [13] and in the external auditory meatus.[14] As Mayer stated, there is a tendency toward new bone formation in the entire petrous bone. Furthermore fragilitas ossium (or osteopsathyrosis) has been repeatedly found associated with otosclerosis [15] (See figures, part III of this paper). It may be concluded, therefore, that the otosclerotic bony changes are a localized dysostosis, in some instances a part of a generalized dysostosis. The hypothesis may be suggested that where the bone-forming processes are "unstable" (Streeter), as in the periotic capsule with its uniquely complicated metamorphosis of cartilage into bone, under the influence of a defective osteogenic gene the bone-producing factors break down seriously. Of course, any breakdown in bone formation in the labyrinthine capsule that leads to deafness is socially an important event, is brought to the physician's attention and adds 1 to the cases of otosclerosis, whereas it might more properly add 1 to the cases of dysostosis.

The first symptoms of otosclerosis are usually noticed between the ages of 10 and 40 years. Table 1 gives the age of onset in our own collection and in that of others.

From table 1 and figure 1 it appears that the commonest age at which deafness is first noticed is between 15 and 20 years, the age at which puberty is well established. The youngest age stated for

10. Guild, S. K. (footnote 9, p. 483).
11. Duel (footnote 4, index of subjects, under "Hyperostoses").
12. Moos, cited by Duel (footnote 4, p. 21).
13. Brühl, cited by Duel (footnote 4, p. 78).
14. Mayer, cited by Duel (footnote 4, p. 210).
15. Duel (footnote 4, index of subjects, under "Fragilitas ossium").

our 316 cases is from 2 to 3 years (twice). There are a few cases in both sexes in which the age of onset is given as 60 or even 65 years. It is always doubtful if in such cases the condition is true otosclerosis. On the other hand, Guild [9] found otosclerotic changes, histologically determined, in a man of 68 years who had not noticed any defect in hearing. It is possible, accordingly, that histologic otosclerotic changes occurring at puberty may go unnoticed until other changes of old age make these slight morphologic defects less tolerable.

The main mass of the distribution of ages "when first noticed" falls between 5 and 45 years (or possibly one should say 35 years). In the still older group the symptoms that have long been in abeyance are doubtless set free by other inciting causes.

TABLE 1.—*Age When Deafness Was "First Noticed"*

	Male Sex					Female Sex					Both Sexes	
				Total					Total			
Age, Years	Right Ear	Left Ear	? Ear	No.	%	Right Ear	Left Ear	? Ear	No.	%	No.	%
Under 5	..	..	..	..	....	..	..	3	3	1.4	3	1.0
5 to 9	1	1	3	5	5.4	4	2	10	16	7.2	21	6.6
10 to 14	2	..	8	10	10.8	10	5	13	28	12.6	38	12.0
15 to 19	4	2	7	13	14.0	10	6	22	38	17.0	51	16.2
20 to 24	1	1	6	8	8.6	10	6	24	40	17.9	48	15.2
25 to 29	3	3	10	16	17.2	11	8	13	32	14.4	48	15.2
30 to 34	2	1	12	15	16.1	7	7	19	33	14.8	48	15.2
35 to 39	..	1	5	6	6.5	1	..	7	8	3.6	14	4.4
40 to 44	1	..	10	11	11.8	3	1	6	10	4.5	21	6.6
45 to 49	..	..	1	1	1.1	..	..	3	3	1.3	4	1.3
50 to 54	..	1	2	3	3.2	..	1	6	7	3.1	10	3.2
Over 55	..	..	5	5	5.4	..	..	5	5	2.3	10	3.2
Totals	14	10	69	93		56	36	131	223		316	
Mean	22.50	27.00	30.40	28.85		22.32	23.89	26.01	24.74		25.95	
Standard deviation	9.25	11.70	13.45	13.05		9.00	9.45	13.35	11.90		12.40	
Probable error of mean	1.67	2.50	1.09	0.91		0.81	1.06	0.79	0.54		0.47	

It is biologically significant that the osteogenic processes, apparently abnormal from embryonic life, should show their defects more prominently at puberty, for puberty is a period of life when osteogenic processes are proceeding with extraordinary velocity. The bones of the leg and arm may increase from 25 to 75 mm. in length in one year and the skull show an unusual growth. Adolescence is a period of widespread change, and many cases of endocrine dysfunction, or disharmony, first become prominent then.

Between the sexes the age of incidence is not very diverse. In the case of the right ear there is no difference. In the case of the left ear the age of onset is possibly slightly later in the male than in the female sex.

3. *Earlier Studies on Inheritance of Otosclerosis.*—That defects of hearing tend to run in families has long been recognized. The classic studies of Alexander Graham Bell on this subject demonstrated the

inheritance of such defects; he showed how defectives tended to marry each other (because they could much more readily converse with each other rather than with normal persons), were in consequence producing a large number of defective offspring and thus were in danger of producing a new race with defective hearing. Other studies have been made on inheritance of defects of hearing by Körner,[16] Hammerschlag,[17] Lundborg,[18] Plate,[19] Albrecht [20] and Bauer and Stein.[21] While Körner, Hammerschlag and Albrecht dealt primarily with inheritance of otosclerosis, Plate and Lundborg were concerned especially with deafmutism. Albrecht concluded that otosclerosis is due to a simple dominant factor, but Bauer and Stein, with larger material and a more complete analysis, reached the conclusion that 2 genetic factors, A and B, are present, which must both be recessive for otosclerosis to appear in the individual.

The conclusions of Bauer and Stein are so significant that it is worth while to consider their methods and results. First, they tabulated for their whole population with defective hearing the diagnosis of the defect in the parents and siblings of patients with otosclerosis. They found that in about 100 families of otosclerotic persons there were 24 cases of otosclerotic parents and 31 of otosclerotic siblings. Moreover, for 23 of the parents there was a diagnosis of labyrinthine hardness of hearing, 3 had chronic inflammation of the inner ear and 5 showed other defects of hearing of an unknown nature. This led the authors to conclude that there is a close relationship between otosclerosis and labyrinthine hardness of hearing and, indeed, their further analysis led them to think that the same genes are responsible for the 2 defects. To judge of the probability of this conclusion being correct, it is necessary to consider the nature of what is known as (to cite its full designation) chronic progressive labyrinthine hardness of hearing. This is differentiated from otosclerosis clinically by the fact that its incidence is usually later in life, by the frequent disturbances of equilibrium and by the so-called positive Rinne test which indicates that perception of tones carried through the air is positively greater than that of tones

16. Körner, O.: Das Wesen der Otosklerose im Lichte der Vererbungslehre, Ztschr. f. Ohrenh. **50**:98, 1905; Arch. Otol. **35**:441, 1906.

17. Hammerschlag, V.: Zur Frage der Vererbbarkeit der Otosklerose, Wien. klin. Rundschau **19**:5, 1905.

18. Lundborg, H.: Ueber die Erblichkeitsverhältnisse der konstitutionellen (hereditären) Taubstummheit, Arch. f. Rassen- u. Gesellsch. Biol. **9**:133, 1912.

19. Plate, L.: Vererbungslehre, Leipzig, Wilhelm Engelmann, 1913.

20. Albrecht, W.: Ueber die Vererbung der konstitutionellen sporadischen Taubstummheit und der Otosklerose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **110**:15, 1922.

21. Bauer, J., and Stein, C.: Vererbung und Konstitution bei Ohrenkrankheiten, Ztschr. f. d. ges. Anat. **10**:483, 1925.

carried through the bones of the head. The last mentioned symptom is in marked contrast with typical otosclerosis, in which the Rinne test is usually negative, which means that sound conductions through the air are not so perfectly perceived as through the bones of the head. Also in labyrinthine deafness there is a narrowing of the upper tone limits, whereas in otosclerosis there is a narrowing of the lower tone limits.

For information as to the anatomic basis of labyrinthine deafness we are especially indebted to Manasse.[22] He has shown that in advanced cases the organ of Corti has become reduced from a columnar epithelium to a pavement epithelium, that even the cochlear nerve has become more or less degenerated and that the nerve fibers are replaced by connective tissue. In respect to the degeneration of the proper hearing nerve, progressive labyrinthine hardness of hearing resembles deaf-mutism in which also the organ of Corti is degenerate. In deaf-mutism, however, the degeneration of the sound-perceiving apparatus occurs very early, frequently before birth, so that properly one should speak less of a degeneration than of a failure to develop, whereas in progressive labyrinthine hardness of hearing there is a secondary deterioration late in life of an organ once well formed and functional. In contrast to progressive labyrinthine hardness of hearing and deaf-mutism, in otosclerosis the acoustic nerve is usually fully functional and shows no signs of degeneration.

A priori, it would seem improbable that a deafness due primarily to a change in the constitution of the bony ear capsule depends on the same genes that cause, in later life, a degeneration of the sound-perceiving apparatus.

To continue with Bauer and Stein, they adopted the hypothesis that otosclerosis is dependent on 1 or more recessive genes. To test this hypothesis they assembled their matings in the following groups: (*a*) mating of 2 normally hearing parents; (*b*) mating of an otosclerotic with a normally hearing parent and (*c*) mating of 2 otosclerotic parents. In determining the proportion of affected offspring it is, on the hypothesis of recessive inheritance, all-essential to make the Weinberg adjustment, and this is done by the "propositus" method. The justification for this method is that the chance of listing a fraternity as otosclerotic is increased as the number of otosclerotic individuals in the fraternity increases. Since most human fraternities are fairly small and many of them comprise only 2 or 3 individuals, many a family will be overlooked that shows no otosclerotic individuals though the genes for otosclerosis are present in the parents. The consequence

22. Manasse, P.: Neue Untersuchungen zur Otosklerosenfrage, Ztschr. f. Ohrenh. **82**:76, 1922.

is that since there has been a selection of just those fraternities that show otosclerosis, the proportion of otosclerotic individuals in the progeny is abnormally high. The propositus method of Weinberg corrects this proportion by cutting out the propositus from consideration as one of the affected individuals and subtracting 1 from the total number of individuals in the fraternity. The incidence of otosclerosis in the fraternity is then computed by the ratio of the remaining otosclerotic individuals in the fraternity to the entire fraternity (from which 1 has been subtracted).[23]

Applying the Weinberg correction, Bauer and Stein found that when both parents have normal hearing, the proportion of otosclerotic offspring (in fraternities in which otosclerosis has been found) is about 6 per cent (or 9 per cent if all sorts of hearing defects in the fraternities are grouped together).

In the case in which one parent is otosclerotic and the other has normal hearing, the proportion of the progeny affected with otosclerosis is about 25 per cent (or 33 per cent with all sorts of defective hearing). Bauer and Stein properly concluded that the 6 per cent defective offspring from the mating of 2 unaffected parents is far from expectation on the hypothesis of a single affected gene. For in that case 25 per cent of the offspring should be affected. Also 26 per cent affected offspring is far from expectation when 1 parent is affected, on the hypothesis of a single affected gene, for in that case 50 per cent of the offspring should be affected. On the other hand, the proportions are what are expected on the assumption of 2 recessive genes, for in that case 6.25 per cent and 25 per cent affected offspring are expected according as there is no affected parent or only 1.

The critical mating to test the hypothesis of double recessive genes in otosclerosis is the mating of 2 otosclerotic parents. As Bauer and Stein have recorded only 2 such matings, their own data do not give a very satisfactory test. They bring into consideration some families cited by others. It appears in these families that a certain proportion of the offspring have normal hearing. This would seem to be rather conclusive evidence against the theory. They have explained the result, however, on 2 possible grounds. Either the unaffected offspring, though they may have reached the ages respectively of 52 and 60 years, may become hard of hearing some time later or the propositus who gave the information about his siblings did not give it correctly—possibly because he did not know the facts. It would appear that either of these 2 explanations proves too much and should affect the statistics

23. The Weinberg method is not without its theoretical difficulties, as Lenz and others have pointed out. However, it affords a welcome partial correction of the results of undue selection of fraternities, and it is easy to apply.

in regard to the progeny of 2 unaffected parents, or of an affected parent mated to a normal one, in such a way as to give proportions very different from expectation on the hypothesis.

Since the proportion of offspring affected with labyrinthine deafness in a fraternity derived from 2 normally hearing parents is not 6 per cent but 10 or 15 per cent, while the proportion of labyrinthine deafness in fraternities in which 1 parent is hard of hearing is about 22 per cent, it is obvious that the hypothesis of 2 recessive genes which fits the case of otosclerosis does not equally well fit the case of labyrinthine deafness.

To explain the larger proportion of persons with labyrinthine deafness, Bauer and Stein have suggested the hypothesis that in labyrinthine deafness 3 recessive genes, instead of 2 pairs in the zygotes, may be sufficient to give the phenotypic condition of deafness, even though the remaining allelomorph is dominant. They assume that the recessive condition becomes dominant. By this time they are quite ready to point out [24] that the conceptions of dominance and recessiveness on which they have hitherto based their conclusions have only a relative significance and are fictions which make it possible to understand and analyze complicated facts.

It will be observed from the foregoing that the hypothesis that otosclerosis depends on 2 recessive factors meets with grave difficulties and is far from satisfactory.

Deaf-mutism is brought by Bauer and Stein into the same general category of inheritance as otosclerosis and labyrinthine deafness, and they seem inclined to conclude that the 3 defects are really due to similar recessive genes and form merely different aspects of the same defect, viz., a developmental inferiority in the organ of hearing.

The conclusions of Bauer and Stein in regard to deaf-mutism differ somewhat from those of Plate.[25] The latter presented a hypothesis that is of great interest to us. He suggested that in addition to a general defective condition, which may be called R, there are 2 dominant conditioning factors, C and K, which must both be present in a homozygous or heterozygous condition in order that there should be deaf-mutism; that is, on this hypothesis, affected persons may have any one of the formulas, RRCCKK, RRCcKK, RRCCKk and RRCcKk.

Bauer and Stein recognize a sex difference in the incidence of otosclerosis and consider for a moment the possibility of a sex-linked inheritance. They point out that in sex-linked dominant inheritance the affected father on the one side has only affected daughters and on the other side (if married to a normally hearing wife) always only normally hearing sons. However, from such matings Bauer and Stein's

24. Bauer and Stein (footnote 21, p. 506).
25. Plate (footnote 19, p. 372).

material shows 11 otosclerotic sons.[26] They, therefore, reject the hypothesis of sex linkage and find the explanation of the fact that females are nearly twice as apt to be affected as males in the various physiologic processes of the female sex life (pregnancy, lactation), which serve as provoking agents to otosclerosis. They do not call attention to the fact that there is no such sex difference in the case of labyrinthine deafness, which, by hypothesis, depends on the same genes and which should show, apparently, the same differential defect in the 2 sexes.

B. MATERIAL AND METHODS

Our material for a study of inheritance of otosclerosis is derived from 2 sources.

The first source is the field studies of Dr. Bess Lloyd Milles who worked with a grant from the Otosclerosis Committee during 1928 and 1929. Dr. Milles was well trained for this field study, from her experiences in field study, her analyses of heredity in other traits and her medical training and contacts. In beginning her work she received great assistance from Drs. Arthur B. Duel, E. B. Dench and T. J. Harris of New York and Dr. F. E. Shambaugh of Chicago. For additional cases aid was sought from, and freely given by, the Volta Bureau. Naturally, a considerable proportion of the propositi of the bureau proved not to be cases of otosclerosis.

After Dr. Milles was obliged to relinquish her work, Miss Lillian Frink took it up in 1930. She made studies of members of the families that Dr. Milles had investigated, in different states of the Mississippi Valley. Later she worked in cooperation with Dr. S. K. Guild of the Otological Research Laboratory, Johns Hopkins Hospital, and studied the family history of a number of otosclerotic patients in and about Baltimore.

During the early part of the study the field worker did 3 things in an interview:

1. She obtained as large and extensive a pedigree as possible from the propositus, and made special inquiry concerning the hearing of each member, or checked this when interviewing other members of the family.

2. She sought special details concerning the members who were hard of hearing. For this purpose the following form was used.

Form B. (Fill out for each person who is hard of hearing)

Name.....Sex.....Age.....Address.....
Which ear is worse?.....At what age was difficulty in hearing first noticed?....
Was there any cause?.....
Did either ear ever discharge?.....Age?.....Your doctor's name?.....
Was there a loss of hearing after each pregnancy?.....
Any headaches?.....How often?.....Any mastoid trouble?.....
Any dizzy spells?.....How often?.....What age?.....
Any noticeable noises in the head?.....What sounds?.....
Any sinus trouble?.....Any nose operation?.....
Were tonsils removed?.....Age?.....
Was the hearing any better after the tonsils were removed?.....
Any sore throat?.....Many colds?.....
Do the teeth decay easily?.....At what age were false teeth necessary?.....
What children's diseases?.....

26. Bauer and Stein (footnote 21, p. 497).

What serious illness? Age?.....
What operations?.....
Was the hearing any better after the operation?.....
Any sign of goiter? Age?.....Was it treated?.....
Ever done any telephone switchboard operating?.....How long?.....
Other suggestions.....
American Otological Society: Committee on Otosclerosis: Eugenic Investigation.
Date of this Record.....

3. She made a short series of anthropometric measurements and observations on as many members of the family as possible and determined whether they were hard of hearing. This schedule included the following items: stature, height of iliac spine, sitting height, height of tibia, length of arm (acromion to stylion), acromion breadth, bicristal diameter, weight, axillary chest girth, width and length of head, bitemporal width, minimum frontal breadth, bizygomatic and bigoniac widths, nasion to stomion, nasion to gnathion, drawing of hand, brittle bones, ocular defects, skin pigmentation, color of eyes and hair and graying of hair. This study was discontinued after January, 1931.

Beginning with January, 1931, Dr. Guild of the Johns Hopkins Hospital offered to cooperate in the study. He furnished the field worker with new cases, including the results of all hearing tests that had been made on the patient, provided access to the history room of the hospital and gave the field worker instructions and tuning forks so that the Rinne, Weber and Schwabach tests might be made in the field.

The cases furnished by Dr. Guild were of 2 types: (1) those provisionally diagnosed as otosclerosis following a clinical examination and (2) those diagnosed as otosclerosis following a histologic examination of sections of ears which had been obtained at autopsies. In the first type of case the procedure was the same as previously (without the anthropometric measurements) with the addition of the question, "Do you hear better in a noisy place, such as a street car or auto?" to form B and the tests mentioned in the preceding paragraph. These tests were performed on the members of the family who were hard of hearing and who had not been examined at Johns Hopkins Hospital. In the second type of case the nearest relative of the propositus was interviewed. Details concerning the hearing of the propositus were particularly sought, and then the same information as in the other cases. Dr. Guild also offered to examine the hearing and give what help and advice he could, free of charge, to relatives of the propositi who would come to the laboratory. The field worker endeavored to get the relatives, especially those who were hard of hearing, to come for the test and made the necessary arrangements. This made it possible to get more accurate information concerning the hearing of relatives.

C. RESULTS

The results of the field work are found in figures 1 to 48 with their associated legends (part III). These pedigrees are, as stated, only a portion of those studied. Among the pedigrees that were obtained through the assistance of the Volta Bureau were some in which it was doubtful if we had to do with otosclerosis. It was finally decided to analyze only those pedigrees in which the diagnosis of otosclerosis had been definitely made in at least 1 individual in the main, or central, fraternity.

It might be urged that the progeny of every mating in which 2 parents had been diagnosed as otosclerotic or not should be described and introduced into the analysis. Obviously, this is impossible for all the cases in which both parents have normal hearing and have only normally hearing offspring. In the cases in which 1 parent has otosclerosis, it sometimes happens that there are no otosclerotic offspring. This may be due to a variety of causes. The progeny may all be young, none having attained the average age of onset of otosclerosis, a considerable proportion of the children may have died young or there may be only 1 or 2 children when expectation calls for an affected child in a fraternity of 2 or 4. Finally, there may be, as has been alleged, cases of noninherited otosclerosis. However, our data do not give any clear picture of noninheritable otosclerosis; and it remains to be shown that the typical symptoms of otosclerosis can arise without the genetic factors that form the constitutional basis for it.

Apart from the grounds of exclusion stated, there has been no conscious selection of the pedigrees studied. In addition, it was decided to examine all available published pedigrees of otosclerosis which were obviously made out with care and were extensive enough to throw light on the problem of the method of inheritance.

In analyzing these pedigrees the entire pedigree was not taken as a unit, since many of these pedigrees comprise several fraternities. Accordingly a particular mating was taken as the unit, and the proportion of affected and unaffected offspring resulting from each mating was considered. Since there is such a striking difference in the incidence of otosclerosis in males and females, the sexes are everywhere kept distinct.

Since the hypothesis finally adopted does not assume recessive factors, it did not seem necessary to use the Weinberg propositus method. Indeed, as will appear in the sequel, the exact proportion of affected individuals in the fraternities is hardly significant for interpreting the method of inheritance.

While, as stated, we have included many of the family histories recorded in the literature, the results obtained through genealogical study by a well trained investigator who goes to the homes, sees as many as possible of the patients' relatives and gets concurrent testimony from (if possible) 2 relatives or acquaintances of those kin who cannot be seen are of a different order of reliability from those pedigree charts compiled in the office from statements made by the patient or relatives who accompany him.

Accordingly much more weight is to be ascribed to our pedigrees than to most of those found in the literature. For the sake of com-

pleteness we have, however, analyzed the pedigrees published by others, and have included them, separately, in this study.

Pedigrees have been published as follows, and those starred (*) have been used in this study:

Albrecht, 1922;* Amadon, 1912;* Bauer and Stein, 1913, 1925,* 1926; Beck, 1910; Bronson, 1917;* Drury, 1926, 1929; and Gottlieb, 1930; Gray, 1912, 1917;* Haike, 1927-1928* 1928;* Hammerschlag, 1905,* 1906,* 1908,* 1909; Heiman, 1909; van der Hoeve and de Kleyn, 1918;* Jenkins, 1927; Kompanejetz, 1930;* Körner, 1905;* Magnus, 1876;* Manasse, 1909; Mayer, 1923; Ruttin, 1922;* Stobie, 1923;* Straat, 1923;* Takahashi, 1924; Terrien, Sainton and Veil, 1927; Undritz, 1928,* and Wojatschek, 1925.*

In our tabulation of results, we have kept apart those fraternities all of whose representatives have reached the age of 30 years, because fraternities that include younger children may be regarded as incomplete, from the standpoint of otosclerosis, as otosclerosis is often first recognized between 30 and 35 years. However, it must be said that not all of the normal persons in the group 30 years of age and over can be said to be outside the bounds of possible incidence of otosclerosis. Leeson,[27] in 1,007 cases, found 41 per cent in which the "incidence" was after 30 years, and in our table 1, 34 per cent of the cases of otosclerosis were first noticed at 30 years or later. A certain proportion of our 30 to 40 year-old siblings of the propositus who are now unaffected may become affected later.

1. *Proportion of the Sexes Affected.*—Since pregnancy increases the symptoms of otosclerosis, it might be regarded as a cause of otosclerosis; if so, the excess of affected females should be much greater after 30 years than before. A count of cases of otosclerosis in the new pedigrees and in those in the literature was made. Among 235 such patients under 30 years of age the proportion of affected females to males was 56:44, or 127 per cent. On the other hand, in persons 30 years of age or over (table 2[28]), the proportion was 61:33 or 185 per cent. That is, the proportion of affected females increases 58 per cent in the later years, leading to the conclusion that the incidence, if not the progress, of otosclerosis is already excessive in the younger females, probably owing to special genetic factors. The later increase may be largely due to the more obvious and emphatic symptoms in the middle-aged women. Pregnancy exaggerates the symptoms of deaf-

27. Leeson, L. A.: The Clinical Aspect of Otosclerosis, J. Laryng. & Otol. **43**:89, 1928.

28. Table 2 alone is considered since the original data as to fraternities are believed to be much more reliable than the data from the literature. For some unknown reason the proportion of males to females in our fraternities is low, viz., 147:189.

TABLE 2.—*Proportion of Sexes Affected with Otosclerosis*

I. Original Cases

A. IN OFFSPRING OF MATINGS IN TABLE

1. All Children in Fraternity 30 Years of Age or Over

Type of Mating	Number with Probable Otosclerosis		Number Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female
Both parents affected....	3	6	0	2	3	8
Mother only affected.....	3	10	3	4	6	14
Father only affected.....	4	15	3	4	7	19
Neither parent affected...	10	8	0	1	10	9
Total.....	20	39	6	11	26	50

2. Children in Fraternity Both Over and Under 30 Years of Age

Type of Mating	(a) Under 30 Years						(b) Over 30 Years					
	Number with Otosclerosis		No. Hard of Hearing (Kind Uncertain)		Total		Number with Otosclerosis		No. Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Both parents affected....	..	..	..	..	..	..	..	..	..	..	..	..
Mother only affected.....	1	1	0	0	1	1	0	0	0	0	0	0
Father only affected.....	..	..	..	..	..	..	..	..	..	..	..	..
Neither parent affected...	1	3	0	0	1	3	3	10	4	1	7	11
Total.....	2	4	0	0	2	4	3	10	4	1	7	11

3. Total Number of Children 30 Years of Age or Over (1 and 2b)

Type of Mating	Number with Probable Otosclerosis		Number Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female
Both parents affected....	3	6	0	2	3	8
Mother only affected.....	3	10	3	4	6	14
Father only affected.....	4	15	3	4	7	19
Neither parent affected...	13	18	4	2	17	20
Total.....	23	49	10	12	33	61

4. Total Number of Children from These Matings (1 and 2)

Type of Mating	Number with Probable Otosclerosis		Number Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female
Both parents affected....	3	6	0	2	3	8
Mother only affected.....	4	11	3	4	7	15
Father only affected.....	4	15	3	4	7	19
Neither parent affected...	14	21	4	2	18	23
Total.....	25	53	10	12	35	65

B. IN ALL FAMILIES CHARTED

Sex	Number with Probable Otosclerosis	Number Hard of Hearing (Kind Uncertain)	Total
Male.....	58	110	168
Female.....	113	98	211
Total.....	171	208	379

TABLE 2.—*Proportion of Sexes Affected with Otosclerosis—Continued*

II. Cases from Literature

A. IN OFFSPRING OF MATINGS IN TABLE

1. All Children in Fraternity 30 Years of Age or Over

Type of Mating	Number with Probable Otosclerosis		Number Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female
Both parents affected....	3	10	0	0	3	10
Mother only affected.....	22	17	0	0	22	17
Father only affected.....	10	17	0	0	10	17
Neither parent affected...	10	17	0	0	10	17
Total.....	45	61	0	0	45	61

2. Children in Fraternity Both Over and Under 30 Years of Age

Type of Mating	(a) Under 30 Years						(b) Over 30 Years					
	Number with Otosclerosis		No. Hard of Hearing (Kind Uncertain)		Total		Number with Otosclerosis		No. Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Both parents affected....	1	0	0	0	1	0	3	3	0	0	3	3
Mother only affected.....	1	1	0	0	1	1	1	5	1	0	2	5
Father only affected.....	0	2	0	0	0	2	4	6	1	0	5	6
Neither parent affected...	1	0	0	0	1	0	10	7	0	0	10	7
Total.....	3	3	0	0	3	3	18	21	2	0	20	21

3. Total Number of Children 30 Years of Age or Over (1 and 2b)

Type of Mating	Number with Probable Otosclerosis		Number Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female
Both parents affected....	6	13	0	0	6	13
Mother only affected.....	23	22	1	0	24	22
Father only affected.....	14	23	1	0	15	23
Neither parent affected...	20	24	0	0	20	24
Total.....	63	82	2	0	65	82

4. Total Number of Children from These Matings (1 and 2)

Type of Mating	Number with Probable Otosclerosis		Number Hard of Hearing (Kind Uncertain)		Total	
	Male	Female	Male	Female	Male	Female
Both parents affected....	7	13	0	0	7	13
Mother only affected.....	24	23	1	0	25	23
Father only affected.....	14	25	1	0	15	25
Neither parent affected...	21	24	0	0	21	24
Total.....	66	85	2	0	68	85

B. IN ALL FAMILIES CHARTED

Sex	Number with Probable Otosclerosis	Number Hard of Hearing (Kind Uncertain)	Total
Male.....	123	13	136
Female.....	163	12	175
Total.....	286	25	311

ness in the otosclerotic patient. The inference may be drawn that the true genetic potentialities of the sexes is first fully shown after 30 years of age.

On the other hand, in our new data the incidence of hardness of hearing other than otosclerosis in the 2 sexes (208 cases) is 43 per cent in the female and 57 per cent in the male (table 2); while the United States census returns for 1900 [29] give for hardness of hearing the female/male ratio of 94 per cent.

From the figures cited, it appears that otosclerosis is peculiar among defects of hearing in showing a striking sex-dimorphism in its incidence. This makes it highly probable that at least 1 of the genes that plays a part in its development is located in the sex chromosome.

2. *Constitution of Otosclerotic Persons.*—It has been repeatedly suggested that otosclerotic persons, as a class, are different in their constitution from the run of the population of their race, sex and age. Bauer and Stein (1914) have particularly developed this point. In their later paper [30] they stated: "Eine regelmässige Bindung der Otosklerose an bestimmte andere Konstitutionsanomalien, sei es der aüsseren Körperform oder bestimmter innerer Organe, nicht nachzuweisen ist. Grosse und kleine, fette und magere, schmal- und breitwüchsige, blonde und brünette Individuen finden sich in unserem Material. [A regular linkage of otosclerosis with certain other constitutional anomalies, whether of the external contour of the body or of certain internal organs is not demonstrable. Persons tall and short, obese and emaciated, narrow and broadly built, blond and brunette, are in our material.]" In order to test this by newer and more critical methods, Dr. Milles, and later Miss Frink, made physical measurements of otosclerotic and nonotosclerotic members of our otosclerotic families. They measured 37 males and 78 females. A special comparison was made between the otosclerotic and nonotosclerotic individuals in families comprising both. There were a number of such families. The number that contained both affected and nonaffected males was too small to study. Fifteen families contained both affected (22) and nonaffected (19) females, and the results of their measurements are given in table 3.

Column 1 in table 3 gives a list of the dimensions measured. Column 2 gives the mean of the measurement of each dimension, based on 78 females, except as otherwise indicated. This is computed from the individual measurements (not grouped into classes). Column 3 gives for the nonotosclerotic persons the average deviation of the individual measurements from the mean of all, as given in column 2;

29. Bell, A. G.: United States Census Returns of 1900 on the Deaf, 1906, p. 99.
30. Bauer and Stein (footnote 21, p. 522).

TABLE 3.—*Comparison of Physical Measurements of Otosclerotic and Nonotosclerotic Females in Fifteen Families*

1	2	3		4		5	6	7	8
		Individuals							
		Normal		Otosclerotic		Difference ÷ Probable Error of Difference	Families Total Deviation		F(N-O)*
Measurement	Average	Average Deviation	Probable Error	Average Deviation	Probable Error		Normal	Otosclerotic	I(N-O)
Stature, mm.	1,578	+20.45	±9.74	−1.33	±10.29	1.54	−4	+15	−0.87
Length of leg, mm.	864	+8.60	±5.97	+1.48	±7.77	0.73	+5	+50	−6.32
Sitting height, mm.	828	+1.78	±5.03	−9.05	±5.18	1.50	+51	−110	+14.87
Height of tibia, mm.	446†	+0.50	±3.28‡	−2.72	±3.74§	0.65	−29#	−30#	+0.31#
Length of arm, mm.	499¶	+0.40	43.14	−5.24	±50.12	0.085	−60	−31	−5.14
Acromion breadth, mm.	363¶	−0.02	±3.50‖	+3.94	±2.06§	0.96	0#	+54#	+13.64#
Bicristal breadth, mm.	319	+2.22	±3.83	−6.28	±4.21	1.49	+68	−77	+17.06
Bicristal breadth : acromion breadth, per cent.	87.9¶	+1.07	±0.88‖	−2.50	±0.95§	2.75	+20.3#	−47.0#	+18.85#
Weight, Kg.	58.97	−0.23	±1.39	−3.45	±1.55	1.55	+5	−37	+13.00
Weight/stature, per cent.	3.74	0.00	±0.08	−0.18	±0.10	1.43	+0.28	−2.35	+14.50
Chest girth, mm.	847°	+2.84	±9.10‖	−12.72	±6.07§	1.42	+103#	−253#	+22.88#
Relative girth, per cent.	53.57°	+0.05	±0.61‖	−0.89	±0.48§	1.21	+11.47#	−16.24#	+29.46#
Length of hand, mm.	166¶	−1.67	±1.16‖	+0.84	±1.45§	1.35	−32#	+2#	+13.55#
Breadth of hand, mm.	80¶	−0.40	±0.62‖	−0.28	±0.64§	0.13	−6#	−5#	+8.33#
Area of hand, sq. mm.	13,487¶	−261.90	±186.93‖	−222.22	±211.44§	0.14	−4,719#	−2,372#	+58.68#
Maximum breadth of head, mm.	142	+0.68	±1.01	−0.66	±1.07	0.91	+8	−13	+15.67
Maximum length of head, mm.	176¶	+2.03	±0.75‖	+3.29	±1.08	0.95	+19	+44	+19.84
Cephalic index, per cent.	80.08¶	+0.24	±0.59‖	−1.32	±0.57	1.90	+3.00	−16.98	+12.81
Bizygomatic breadth, mm.	115	0.00	±1.28	−1.98	±1.55	0.99	−3	−25	+11.11
Bizygomatic breadth : maximum breadth, per cent.	80.8	+0.46	±0.67	−1.98	±1.00	2.03	−6.1	−11.8	+2.34
Bigonial breadth, mm.	92	+0.46	±0.88	−1.98	±1.00	1.83	+11	−28	+15.98
Bigonial breadth : maximum breadth, per cent.	65.0	−0.18	±0.64	−2.00	±0.85	1.72	+1.5	−23.1	+13.52
Stomion to gnathion, mm.	41	−0.64	±0.76	+1.32	±0.67	1.94	−11	+12	+11.73

* Quotient of the difference between the entries of columns 6 and 7, divided by the difference between the entries of columns 3 and 4.
† Seventy-six individuals.
‡ Twenty individuals.
§ Eighteen individuals.
# Fourteen families.
¶ Seventy-seven individuals
‖ Twenty-one individuals.
° Seventy-four individuals.

also the probable errors of this mean. Since the average deviation is computed from a selected (normal) fraction of the 78 females in column 2, it departs sometimes widely from 0. Column 4 gives for the otosclerotic group the average deviation with probable errors obtained in the same way as those in column 3. Column 5 gives the ratio of the algebraic difference between the average deviation of normal and otosclerotic persons to the probable error of the difference. Column 6 gives the sum of all deviations from the mean (column 1) of the means of the nonotosclerotic females in each of the 15 families containing both nonotosclerotic and otosclerotic females. Similarly, column 7 gives the sum of all deviations from the mean (column 2) of otosclerotic females in each of the same 15 families. Since 15 families are involved, these sums are, in the long run, 15 times larger than the average deviation of 1 family. Column 8 gives the ratio of the difference between the entries of columns 6 and 7 divided by the difference between the entries of columns 3 and 4. Since the data of columns 6 and 7 are *sums* for 15 families and the data of columns 3 and 4 are *averages* of individuals, one might expect the quotients of column 8 to have the value of about 15, and they are seen to fluctuate about that value.

Conclusions: From table 3 the following conclusions are drawn. In the female sex otosclerotic persons differ from unaffected persons in the ratio: pelvic (bicristal) breadth/shoulder (acromion) breadth. This is exceptionally small in the otosclerotic group. The small ratio in the latter is due partly to broad shoulders and partly to narrow pelvic (bicristal) breadth. This ratio is much smaller in the average male (77.4 per cent) than in the average female (87.9 per cent). Its smallness in the otosclerotic female is an approach toward the male type.

As for chest girth in the 15 selected families, there was a heavy run of slender persons among the otosclerotic group, while those in the nonotosclerotic group were of average size. In the average individual series the otosclerotic persons were only slightly under expectation, so that the quotient would be 15, if the families are fair samples of the population of 78 females. The somewhat refined test of the "families" suggests that otosclerotic females in a family are slenderer than the nonotosclerotic females in that family. In relative girth, also, the otosclerotic group is slenderer than the nonotosclerotic.

As for the other dimensions, there is no clear difference in either sex between nonotosclerotic and otosclerotic persons. In constitution they are not strikingly dissimilar.

3. *Familial Distribution in Affected Families.*—In table 4 are given for our original families the number and proportion of affected sons and daughters, the progeny of each mating of the 4 types: (1) both

TABLE 4.—*Number and Proportion of Affected Sons and Daughters in Original Family Histories*

Family History Parents	Sex	Number Children	Probably Otosclerotic	Hard of Hearing (Kind Uncertain)	Comment*
1. BOTH PARENTS AFFECTED					
(a) Children 30 Years of Age or Over					
F. H. 12	Male.....	3	2	0	Male: died at 46 years
I 8-9	Female...	3	2	1	
	Total....	6	4	1	
F. H. 17	Male.....	2	1	0	Male: died at 63 years
II 8-9	Female...	5	4	1	
	Total....	7	5	1	
				Total Number Hard of Hearing	Probably Otosclerotic, per Cent
No. of fraternities, 2	Male.....	5	3	0	0 60 0 60
	Female...	8	6	2	8 75 25 100
	Total....	13	9	2	11 69 15 85
(b) Children Both Over and Under 30 Years of Age					
	No cases				
(c) Children Under 30 Years of Age					
F. H. 22	Male.....	0	0	0	
	Female...	1	1	0	
	Total....	1	1	0	
2. MOTHER ONLY AFFECTED					
(a) Children 30 Years of Age or Over					
F. H. 1	Male.....	2	1	0	Male: 54 years
II 2-1	Female...	3	2	0	Female: 40 years
	Total....	5	3	0	
F. H. 5	Male.....	1	0	1	
II 6-7	Female...	3	2	0	Female: 44 years
	Total....	4	2	1	
F. H. 21	Male.....	2	0	0	
II 7-8	Female...	2	0	1	Female: 46 years
	Total....	4	0	1	
F. H. 25	Male.....	0	0	0	
III 4-5	Female...	2	1	0	Female: 32 years
	Total....	2	1	0	
F. H. 27	Male.....	0	0	0	
II 7-6	Female...	1	1	0	
	Total....	1	1	0	
F. H. 29	Male.....	2	0	0	
†II 5-6	Female...	1	1	0	
	Total....	3	1	0	
F. H. 30	Male.....	1	0	1	
II 15-14	Female...	2	1	0	
	Total....	3	1	1	
F. H. 34	Male.....	2	0	1	
†II 12-13	Female...	3	1	2	
	Total....	5	1	3	
F. H. 41	Male.....	1	1	0	
†II 1-2	Female...	0	0	0	
	Total....	1	1	0	
F. H. 46	Male.....	3	1	0	Male: died at 30 years
IV 2-1	Female...	5	0	1	Female: 75, 67, 54 years
					Female: died at 74 years
	Total....	8	1	1	
F. H. 48	Male.....	1	0	0	Male: died at 44 years
II 5-4	Female...	2	1	0	Female: 57 years
	Total....	3	1	0	
No. of fraternities, 11	Male.....	15	3	3	6 20 20 40
	Female...	24	10	4	14 42 17 58
	Total....	39	13	7	20 33 18 51

* Unless otherwise stated, the data under "Comment" refer to the age of unaffected individuals.

TABLE 4.—*Number and Proportion of Affected Sons and Daughters in Original Family Histories—Continued*

MOTHER ONLY AFFECTED—Continued

(b) Children Both Over and Under 30 Years of Age

Family History Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment
		Children Under 30 Years of Age			
F. H. 37 †II 7-8	Male.....	1	1	0	
	Female...	0	0	0	
	Total....	1	1	0	
F. H. 43 II 5-6	Male.....	0	0	0	Female: 26 years
	Female...	2	1	0	
	Total....	2	1	0	

					Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
No. of fraternities, 2	Male.....	1	1	0	1	100	0	100
	Female...	2	1	0	1	50	0	50
	Total....	3	2	0	2	67	0	67
		Children 30 Years of Age or Over						
F. H. 37 †II 7-8	Male.....	3	0	0	Male: 38, 35, 30 years			
	Female...	2	0	0	Female: 32, 32 years			
	Total....	5	0	0				
F. H. 43 II 5-6	Male.....	0	0	0				
	Female...	2	0	0	Female: 38, 35 years			
	Total....	2	0	0				
No. of fraternities, 2	Male.....	3	0	0	..	..	..	0
	Female...	4	0	0	..	..	..	0
	Total....	7	0	0	..	..	..	0

3. FATHER ONLY AFFECTED: FAMILIES WITH AFFECTED CHILDREN

(a) Children 30 Years of Age or Over

Family History Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Family History Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)
F. H. 2 †III 5-6	Male...	2	0	1	F. H. 28 III 6-7	Male...	0	0	0
	Female	3	2	0		Female	1	1	0
	Total..	5	2	1		Total..	1	1	0
F. H. 8 III 30 and IV 3	Male...	1	1	0	F. H. 29 II 2-1	Male...	4	1	0
	Female	3	3	0		Female	5	0	0
	Total..	4	4	0		Total..	9	1	0
F. H. 8 III 4 and II 32	Male...	2	1	0	F. H. 33 †II 6-7	Male...	2	0	0a
	Female	3	2	0		Female	4	2	2
	Total..	5	3	0		Total..	6	2	2
F. H. 8 III 32-31	Male...	2	1	0	F. H. 36 †III 11-10	Male...	1	0	1
	Female	3	0	0		Female	2	1	1
	Total..	5	1	0		Total..	3	1	2
F. H. 14 †II 5-6	Male...	3	0	0	F. H. 38 †II 6-7	Male...	2	0	0
	Female	7	1	0		Female	4	1	0
	Total..	10	1	0		Total..	6	1	0
F. H. 26 †II 7-8	Male...	2	0	1b					
	Female	4	2	1c					
	Total..	6	2	2					

					Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
No. of fraternities, 11	Male...	21	4	3	7	19	14	33
	Female	39	15	4	19	38	10	49
	Total..	60	19	7	26	32	12	43

(b) Children Both Over and Under 30 Years of Age

No cases

† The diagnosis of the affected person is somewhat uncertain.
a. Unaffected sons, aged 40 and 38.
b. Unaffected son, aged 54.
c. Unaffected daughter, aged 60.

TABLE 4.—*Number and Proportion of Affected Sons and Daughters in Original Family Histories—Continued*

4. NEITHER PARENT AFFECTED: FAMILIES WITH AFFECTED CHILDREN

(a) Children 30 Years of Age or Over

Family History Parents	Sex	Number Children	Probably Otosclerotic	Hard of Hearing (Kind Uncertain)	Family History Parents	Sex	Number Children	Probably Otosclerotic	Hard of Hearing (Kind Uncertain)
F. H. 3	Male...	1	1	0	F. H. 20	Male...	1	0	0
I 7 and	Female	0	0	0	II 1-2	Female	1	1	0
II 10	Total..	1	1	0		Total..	2	1	0
F. H. 6	Male...	1	1	0	F. H. 23	Male...	0	0	0
III 21-22	Female	0	0	0	II 1-2	Female	1	1	0
	Total..	1	1	0		Total..	1	1	0
F. H. 8	Male...	1	1	0	F. H. 32	Male...	1	1	0
II 28-29	Female	1	0	0	III 14-15	Female	1	0	0
	Total..	2	1	0		Total..	2	1	0
F. H. 9	Male...	0	0	0	F. H. 39	Male...	2	0	0d
II 3-4	Female	1	1	0	II 3-4	Female	1	1	0
	Total..	1	1	0		Total..	3	1	0
F. H. 10	Male...	4	3	0e	F. H. 42	Male...	0	0	0
II 3-4	Female	1	0	0f	II 7-8	Female	1	1	0
	Total..	5	3	0		Total..	1	1	0
F. H. 13	Male...	1	0	0g	F. H. 44	Male...	2	1	0h
II 3-4	Female	1	1	0	II 13-14	Female	0	0	0
	Total..	2	1	0		Total..	2	1	0
F. H. 19	Male...	3	2	0i	F. H. 46	Male...	3	0	0j
II 8-9	Female	2	1	0	V 18-19	Female	4	1	1
	Total..	5	3	0k		Total..	7	1	1

					Total Number Hard of Hearing	Probably Otosclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
No. of fraternities, 14	Male...	20	10	0	10	50	0	50
	Female	15	8	1	9	53	7	60
	Total..	35	18	1	19	51	3	54

(b) Children Both Over and Under 30 Years of Age

		Children Under 30 Years of Age			Children 30 Years of Age and Over		
Family History Parents	Sex	Number Children	Probably Otosclerotic	Hard of Hearing (Kind Uncertain)	Number Children	Probably Otosclerotic	Hard of Hearing (Kind Uncertain)
F. H. 4	Male.....	0	0	0	3	0	1l
II 3-4	Female.....	4	0	0m	3	2	0n
	Total.....	4	0	0	6	2	1
F. H. 7	Male.....	1	1	0	2	0	1
II 2-3	Female.....	0	0	0	2	1	0
	Total.....	1	1	0	4	1	1

d. Unaffected sons, aged 42 and 40.
e. Unaffected son, aged 40.
f. Unaffected daughter, aged 34.
g. Unaffected son, aged 38.
h. Unaffected son, aged 31.
i. Unaffected son, aged 58.
j. Son who was hard of hearing in connection with his final illness; he was considered not affected.
k. Unaffected daughter who died at 56 years of age.
l. One son had hardness of hearing which was not otosclerosis; he was deaf in the left ear from cerebrospinal meningitis, which left him paralyzed. He died at the age of 31. The two unaffected sons were 41 and 32 years of age.
m. Unaffected daughters, aged 22, 29, 27 and 24 years.
n. Unaffected daughter, aged 37.

TABLE 4.—*Number and Proportion of Affected Sons and Daughters in Original Family Histories—Continued*

NEITHER PARENT AFFECTED: FAMILIES WITH AFFECTED CHILDREN—Continued

(b) Children Both Over and Under 30 Years of Age—Continued

Family History Parents	Sex	Children Under 30 Years of Age: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Children 30 Years of Age and Over: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)
F. H. 11	Male	1	0	0	2	0	0
III 3-4	Female	2	1	0	0	0	0
	Total	3	1	0	2	0	0
F. H. 18	Male	1	0	0	2	0	0
II 3-4	Female	2	2	0	2	0	0
	Total	3	2	0	4	0	0
F. H. 35	Male	0	0	0	3	0	0o
II 1-2	Female	1	0	0p	1	1	0
	Total	1	0	0	4	1	0
F. H. 40	Male	1	0	0q	2	1	0
II 10-11	Female	0	0	0	4	0	0r
	Total	1	0	0	6	1	0
F. H. 44	Male	0	0	0	3	0	1s
II 3-4	Female	1	0	0t	3	2	0u
	Total	1	0	0	6	2	1
F. H. 45	Male	0	0	0	4	2	1
II 4-5	Female	1	0	0v	2	1	1
	Total	1	0	0	6	3	2
F. H. 46	Male	1	0	0w	1	0	0x
V 12 and	Female	0	0	0	3	2	0y
VI 16	Total	1	0	0	4	2	0
F. H. 47	Male	0	0	0	0	0	0
II 5-6	Female	1	0	0z	3	1	0
	Total	1	0	0	3	1	0
No. of fraternities, 10	Male	5	1	0	22	3	4
	Female	12	3	0	23	10	1
	Total	17	4	0	45	13	5

Family History Parents	Sex	Children Under 30 Years of Age: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing	Children 30 Years of Age and Over: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
No. of fraternities, 10	Male	1	20	0	20	7	14	18	32
	Female	3	25	0	25	11	43	4	48
	Total	4	24	0	24	18	29	11	40

o. Unaffected sons, aged 39, 33 and 32.
p. Unaffected daughter, aged 29.
q. Unaffected son, aged 28.
r. Unaffected daughters, aged 43, 37, 33 and 30.
s. Unaffected sons, aged 43 and 37.
t. Unaffected daughter, aged 18.
u. Unaffected daughter, aged 46.
v. Unaffected daughter, who died at 8 years of age.
w. Unaffected son, who died at 18 years of age.
x. Unaffected son, aged 37.
y. Unaffected daughter, aged 35.
z. Unaffected daughter, who died at 18 years of age.

TABLE 5.—*Number and Proportion of Affected Sons and Daughters in Pedigrees from the Literature*

1. BOTH PARENTS AFFECTED

(a) Children 30 Years of Age or Over

Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment
Hammerschlag, 1905, table II II 7 and III 2	Male.....	2	2	0	
	Female...	5	5	0	
	Total....	7	7	0	
Haike, 1927-1928, ped. 8a IV 35 and 36	Male.....	0	0	0	
	Female...	1	1	0	
	Total....	1	1	0	
Bauer and Stein, 1925, chart 6 I 1 and 2	Male.....	2	1	0	Male: 52 years
	Female...	5	4	0	Female: 60 years
	Total....	7	5	0	

		Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
No. of fraternities, 3	Male.....	3	75	0	75
	Female...	10	91	0	91
	Total....	13	87	0	87

(b) Children Both Over and Under 30 Years of Age

		Children Under 30 Years of Age			Children 30 Years of Age and Over		
Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)
Gray, 1917, table III II 3 and 7	Male.....	1	1	0	2	1	0a
	Female.....	0	0	0	3	1	0b
	Total.....	1	1	0	5	2	0
Gray, 1917, table VIII II 1 and 2	Male.....	1	0	0c	2	2	0
	Female.....	0	0	0	5	2	0d
	Total.....	1	0	0	7	4	0
No. of fraternities, 2	Male.....	2	1	0	4	3	0
	Female.....	0	0	0	8	3	0
	Total.....	2	1	0	12	6	0

	Children Under 30 Years of Age				Children 30 Years of Age and Over			
Sex	Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing	Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
Male.....	1	50	0	50	3	75	0	75
Female.....	0	..	..	..	3	38	0	38
Total.....	1	50	0	50	6	50	0	50

a. Unaffected son, aged 37 years.
b. Unaffected daughters, aged 33 and 30 years.
c. Unaffected son, who died in childhood, before the age at which otosclerosis is likely to appear.
d. Unaffected daughters, aged 47 and 53 years.

TABLE 5.—*Number and Proportion of Affected Sons and Daughters in Pedigrees from the Literature—Continued*

2. MOTHER ONLY AFFECTED. FAMILIES WITH AFFECTED CHILDREN

(a) Children 30 Years of Age or Over

Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment
Hammerschlag, 1905, table II I 2	Male.....	4	3	0	One son is omitted, as nothing is known about his hearing
	Female...	2	1	0	
	Total....	6	4	0	
Körner, 1905, II †II 1	Male.....	4	2	0	
	Female...	5	3	0	
	Total....	9	5	0	
Amadon, 1912, p. 927 †I 2	Male.....	2	2	0	
	Female...	3	3	0	
	Total....	5	5	0	
Amadon, 1912, p. 932 I 2	Male.....	1	1	0	
	Female...	0	0	0	
	Unknown	6	5	0	
	Total....	7	6	0	
Wojatschek, 1925, table IV-1 II 3	Male.....	5	3	0	
	Female...	2	0	0	
	Total....	7	3	0	
Wojatschek, 1925, table 7, pt. 14 I 1	Male.....	0	0	0	
	Female...	3	2	0	
	Total....	3	2	0	
Wojatschek, 1925, table 7, pt. 19 III 2	Male.....	1	1	0	
	Female...	1	0	0	
	Total....	2	1	0	
Straat, 1923 I 2	Male.....	1	1	0	
	Female...	3	1	0	
	Total....	4	2	0	
Van der Hoeve and de Kleyn, 1918, family X I 1	Male.....	1	1	0	
	Female...	5	3	0	One unaffected daughter, aged 49; other died at age of 46
	Total....	6	4	0	
Gray, 1917, table XIV †I 4	Male.....	1	1	0	
	Female...	1	1	0	
	Unknown	3	3	0	
	Total....	5	5	0	
Haike, 1927-1928, ped. 8a II 1	Male.....	6	1	0	Unaffected sons, aged 88 and 82
	Female...	3	0	0	Unaffected daughters, aged 92 and 86
	Total....	9	1	0	
Haike, 1928, ped Be II 3	Male.....	8	4	0	
	Female...	1	0	0	
	Total....	9	4	0	
Haike, 1928 ped. Be IV 11	Male.....	3	2	0	
	Female...	1	1	0	
	Total....	4	3	0	
Bronson, 1917, chart 1 II 1	Male.....	0	0	0	
	Female...	3	2	0	
	Total....	3	2	0	
No. of fraternities, 14	Male.....	37	22	0	
	Female...	33	17	0	
	Unknown	9	8	0	
	Total....	79	47	0	

Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
22	59	0	59
17	52	0	52
8	89	0	89
47	59	0	59

† The diagnosis of the affected person is somewhat uncertain.

TABLE 5.—*Number and Proportion of Affected Sons and Daughters in Pedigrees from the Literature—Continued*

MOTHER ONLY AFFECTED—Continued

(b) Children Both Over and Under 30 Years of Age

Reference Parents	Sex	Children Under 30 Years of Age: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Children 30 Years of Age and Over: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)
Haike, 1927-1928, ped. Ne II 2	Male	0	0	0	0	0	0
	Female	1	0	0e	1	1	0
	Total	1	0	0	1	1	0
Gray, 1917, table VI I 2	Male	2	1	0f	1	0	0
	Female	0	0	0	3	2	0
	Total	2	1	0	4	2	0
Körner, 1905, I III 6	Male	1	0	0g	0	0	0
	Female	1	1	0	3	1	0h
	Total	2	1	0	3	1	0
Körner, 1905, III II 1	Male	1	0	0i	1	1	0
	Female	1	0	0i	0	0	0
	Total	2	0	0	1	1	0
Haike, 1928, ped. Be III 15	Male	1	0	0j	1	0	1
	Female	0	0	0	1	1	1
	Total	1	0	0	2	1	1
No. of fraternities, 5	Male	5	1	0	3	1	1
	Female	3	1	0	8	5	0
	Total	8	2	0	11	6	1

Sex	Children Under 30 Years of Age: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing	Children 30 Years of Age and Over: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
Male	1	20	0	20	2	33	33	67
Female	1	33	0	33	5	63	0	63
Total	2	25	0	25	7	55	9	64

3. FATHER ONLY AFFECTED: FAMILIES WITH AFFECTED CHILDREN

(a) Children 30 Years of Age and Over *

Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment
Hammerschlag, 1905, table I II 3	Male	2	1	0	
	Female	2	1	0	
	Total	4	2	0	
Hammerschlag, 1905, table II II 3	Male	1	1	0	
	Female	1	1	0	
	Unknown	8	0	0	
	Total	10	2	0	
Körner, 1905, I II 7	Male	2	2	0	
	Female	3	1	0	
	Total	5	3	0	
Haike, 1928, ped. Be II 11	Male	3	1	0	
	Female	1	1	0	
	Total	4	2	0	

* Some children may have been under 30 years of age, but in cases in which the ages were not given, the children were assumed to be 30 years old or more.

e. Unaffected daughter, who died at the age of 22.
f. Unaffected son, aged 21.
g. Unaffected son, aged 25.
h. Unaffected daughters, aged 30 and 33.
i. Twins, aged 22.
j. Son, who died at the age of 23.

TABLE 5.—*Number and Proportion of Affected Sons and Daughters in Pedigrees from the Literature—Continued*

FATHER ONLY AFFECTED—Continued

(a) Children 30 Years of Age and Over—Continued

Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment
Wojatschek, 1925, table IV-2 I 2	Male.....	2	1	0	
	Female...	4	3	0	
	Total....	6	4	0	
Wojatschek, 1925, table 7, pt. 18 I 2	Male.....	1	0	0	
	Female...	2	2	0	
	Total....	3	2	0	
Gray, 1917, table XIII III 5	Male.....	4	1	0	
	Female...	6	4	0	
	Unknown	1	0	0	
	Total....	11	5	0	
Magnus, 1876 †I 1	Male.....	0	0	0	
	Female...	1	1	0	
	Unknown	12	6	0	
	Total....	13	7	0	
Undritz, 1928, no. 34	Male.....	3	1	0	
	Female...	1	0	0	
	Total....	4	1	0	
Wojatschek, 1925, table 7, pt. 17 †III 3	Male.....	2	1	0	
	Female...	0	0	0	
	Total....	2	1	0	
Wojatschek, 1925, table 7, pt. 19 †II 1	Male.....	2	1	0	
	Female...	2	2	0	
	Total....	4	3	0	
Gray, 1917, table XI †III 2	Male.....	0	0	0	
	Female...	1	1	0	
	Total....	1	1	0	
No. of fraternities, 12	Male.....	22	10	0	
	Female...	24	17	0	
	Unknown	21	6	0	
	Total....	67	33	0	

	Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
	10	45	0	45
	17	71	0	71
	6	29	0	29
	33	49	0	49

(b) Children Both Over and Under 30 Years of Age

Reference Parents	Sex	Children Under 30 Years of Age: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Children 30 Years of Age and Over: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)
Ruttin, 1922, family S †II 3	Male.....	0	0	0	3	2	1
	Female.....	2	0	0k	2	2	0
	Total.....	2	0	0	5	4	1
Ruttin, 1922, family N II 4	Male.....	0	0	0	0	0	0
	Female.....	1	1	0	1	1	0
	Total.....	1	1	0	1	1	0
Stobie, 1923, fig. 1 II 4	Male.....	1	0	0l	4	2	0
	Female.....	1	1	0	2	2	0
	Total.....	2	1	0	6	4	0
Gray, 1917, table XI 2	Male.....	0	0	0	1	0	0
	Female.....	1	0	0m	3	1	0
	Unknown.....	0	0	0	1	1	0
	Total.....	1	0	0	5	2	0
No. of fraternities, 4	Male.....	1	0	0	8	4	1
	Female.....	5	2	0	8	6	0
	Unknown.....	0	0	0	1	1	0
	Total.....	6	2	0	17	11	1

† The diagnosis of the affected person is somewhat uncertain.
k. One unaffected daughter, aged 15; other died at 29 years of age
l. Unaffected son, who died at the age of 4 years.
m. Unaffected daughter, who died young.

TABLE 5.—*Number and Proportion of Affected Sons and Daughters in Pedigrees from the Literature—Continued*

FATHER ONLY AFFECTED—Continued

(b) Children Both Over and Under 30 Years of Age—Continued

Sex	Children Under 30 Years of Age: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing	Children 30 Years of Age and Over: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
Male	0	0	0	0	5	50	13	63
Female	2	40	0	40	6	75	0	75
Unknown	0	..	..	..	1	100	0	100
Total	2	33	0	33	12	65	6	71

4. NEITHER PARENT AFFECTED: FAMILIES WITH AFFECTED CHILDREN

(a) Children 30 Years of Age or Over

Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment
Albrecht, 1922, case 3 II 1 and 2	Male	1	1	0	
	Female	0	0	0	
	Unknown	7	0	0	
	Total	8	1	0	
Gray, 1917, table V II 1 and 2	Male	3	3	0	
	Female	3	1	0	Unaffected daughters, aged 45 and 46
	Total	6	4	0	
Gray, 1917, table VII	Male	3	2	0	
	Female	8	2	0	Unaffected daughters, from 50 to 33 years of age
	Total	11	4	0	
Gray, 1917, table XIII II 1	Male	1	1	0	
	Female	3	1	0	
	Total	4	2	0	
Gray, 1917, table XIII II 2	Male	1	1	0	
	Female	0	0	0	
	Total	1	1	0	
Gray, 1917, table XIII II 5	Male	0	0	0	
	Female	1	1	0	
	Total	1	1	0	
Gray, 1917, table XV III 2 and 5	Male	3	0	0	
	Female	7	3	0	
	Total	10	3	0	
Haike, 1927-1928, ped. 8a III 2	Male	4	2	0	
	Female	6	0	0	
	Total	10	2	0	
Heike, 1927-1928, ped. 8a III 9	Male	1	0	0	
	Female	3	3	0	
	Total	4	3	0	
Haike, 1927-1928, ped. 8a III 19	Male	0	0	0	
	Female	2	1	0	
	Total	2	1	0	
Haike, 1928, Ma II 4	Male	0	0	0	
	Female	1	1	0	
	Unknown	7	0	0	Unaffected children of unknown sex, aged from 50 to 30
	Total	8	1	0	
Haike, 1928, ped. Ri II 3	Male	4	0	0	
	Female	4	1	0	
	Total	8	1	0	
Heike, 1928, ped. Ri II 6 and 7	Male	1	0	0	
	Female	3	1	0	
	Total	4	1	0	

TABLE 5.—*Number and Proportion of Affected Sons and Daughters in Pedigrees from the Literature—Continued*

NEITHER PARENT AFFECTED—Continued

(a) Children 30 Years of Age or Over—Continued

Reference Parents	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Comment			
Haike, 1928, ped. Be II 15 and 16	Male.....	4	0	0				
	Female...	2	1	0				
	Total....	6	1	0				
Undritz, 1928, no. 38 II 1 and 2	Male.....	0	0	0				
	Female...	1	1	0	Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
	Total....	1	1	0				
No. of fraternities, 15	Male.....	26	10	0	10	38	0	38
	Female...	44	17	0	17	39	0	39
	Unknown	14	0	0	0	0	0	0
	Total....	84	27	0	27	32	0	32

(b) Children Both Over and Under 30 Years of Age

Reference Parents	Sex	Children Under 30 Years of Age: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Children 30 Years of Age and Over: Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)
Gray, 1917, table 2 III 4 and 11	Male.....	0	0	0	5	3	0n
	Female.....	1	0	0o	3	3	0
	Total.....	1	0	0	8	6	0
Haike, 1928, ped. Ma II 2 and III 6	Male.....	0	0	0	4	2	0
	Female.....	1	0	0p	4	1	0
	Total.....	1	0	0	8	3	0
Haike, 1928, ped. Co II 4 and 5	Male.....	1	0	0	1	1	0
	Female.....	2	0	0	0	0	0
	Total.....	3	0	0	1	1	0
Haike, 1928, ped. Th III 3 and 4	Male.....	0	0	0	1	1	0
	Female.....	0	0	0	5	1	0
	Unknown.....	3	0	0	0	0	0
	Total.....	3	0	0	6	2	0
Hammerschlag, 1906, fig. II III 2	Male.....	4	0	0	1	1	0
	Female.....	0	0	0	4	2	0
	Total.....	4	0	0	5	3	0
Hammerschlag, 1908, ped. IX III 1	Male.....	3	0	0	3	1	0
	Female.....	1	0	0	2	0	0
	Unknown.....	0	0	0	1	0	0
	Total.....	4	0	0	6	1	0
Kompanejetz, 1930, cases 1 and 2 II 1 and 2	Male.....	3	1	0	1	1	0
	Female.....	1	0	0	0	0	0
	Total.....	4	1	0	1	1	0
No. of fraternities, 7	Male.....	11	1	0	16	10	0
	Female.....	6	0	0	18	7	0
	Unknown.....	3	0	0	1	0	0
	Total.....	20	1	0	35	17	0

Sex	Children Under 30 Years of Age: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing	Children 30 Years of Age and Over: Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
Male.....	1	9	0	9	10	63	0	63
Female.....	0	0	0	0	7	39	0	39
Unknown.....	0	0	0	0	0	0	0	0
Total.....	1	5	0	5	17	49	0	49

n. Unaffected sons, aged 30 and 41.
o. Unaffected daughter, aged 25.
p. Unaffected daughter, who died at 9 years of age.

TABLE 6.—*Summary of Actual Findings in Fraternities Containing Affected Children*

1. BOTH PARENTS AFFECTED *

(a) Offspring 30 Years of Age and Over

	Number in Fraternity	Sex	Number Children	Probably Oto-sclerotic	Hard of Hearing (Kind Uncertain)	Total Number Hard of Hearing	Probably Oto-sclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
Original.....	2	Male.....	5	3	0	3	60	0	60
		Female...	8	6	2	8	75	25	100
Literature....	3	Male.....	4	3	0	3	75	0	75
		Female...	11	10	0	10	91	0	91
Total.....	5	Male.....	9	6	0	6	67	0	67
		Female...	19	16	2	18	84	11	95
		Total....	28	22	2	24	79	7	86

(b) Offspring Both Over and Under 30 Years of Age

(1) Under 30 Years

Original.....	0	Male.....	..	..	..	..	..	..	..
		Female...	..	..	..	..	..	..	..
Literature....	2	Male.....	2	1	0	1	50	0	50
		Female...	0	0	0	0	..	..	..
		Total....	2	1	0	1	50	0	50

(2) 30 Years and Over

Original.....	0	Male.....	..	..	..	..	..	..	..
		Female...	..	..	..	..	..	..	..
Literature....	2	Male.....	4	3	0	3	75	0	75
		Female...	8	3	0	3	38	0	38
		Total....	12	6	0	6	50	0	50

2. MOTHER ONLY AFFECTED

(a) Offspring 30 Years of Age and Over

Original.....	11	Male.....	15	3	3	6	20	20	40
		Female...	24	10	4	14	42	17	58
Literature....	14	Male.....	37	22	0	22	59	0	59
		Female...	33	17	0	17	52	0	52
		Unknown	9	8	0	8	89	0	89
Total.....	25	Male.....	52	25	3	28	48	6	54
		Female...	57	27	4	31	47	7	54
		Unknown	9	8	0	8	89	0	89
		Total....	118	60	7	67	51	6	57

(b) Offspring Both Over and Under 30 Years of Age

(1) Under 30 Years

Original.....	2	Male.....	1	1	0	1	100	0	100
		Female...	2	1	0	1	50	0	50
Literature....	5	Male.....	5	1	0	1	20	0	20
		Female...	3	1	0	1	33	0	33
Total.....	7	Male.....	6	2	0	2	33	0	33
		Female...	5	2	0	2	40	0	40
		Total....	11	4	0	4	36	0	36

(2) 30 Years and Over

Original.....	2	Male.....	3	0	0	0	0	0	0
		Female...	4	0	0	0	0	0	0
Literature....	5	Male.....	3	1	1	2	33	33	67
		Female...	8	5	0	5	63	0	63
Total.....	7	Male.....	6	1	1	2	17	17	33
		Female...	12	5	0	5	42	0	42
		Total....	18	6	1	7	33	6	39

* Omitting table 4, 1c.

TABLE 6.—*Summary of Actual Findings in Fraternities Containing Affected Children—Continued*

	Number in Fraternity	Sex	Number Children	Probably Otosclerotic	Hard of Hearing (Kind Uncertain)	Total Number Hard of Hearing	Probably Otosclerotic, per Cent	Uncertain, per Cent	Total per Cent Hard of Hearing
3. FATHER ONLY AFFECTED									
(a) Offspring 30 Years of Age and Over									
Original.....	11	Male.....	21	4	3	7	19	14	33
		Female...	39	15	4	19	38	10	49
Literature....	12	Male.....	22	10	0	10	45	0	45
		Female...	24	17	0	17	71	0	71
		Unknown	21	6	0	6	29	0	29
Total.....	23	Male.....	43	14	3	17	33	7	40
		Female...	63	32	4	36	51	6	57
		Unknown	21	6	0	6	29	0	29
		Total....	127	52	7	59	41	6	46
(b) Offspring Both Over and Under 30 Years of Age									
(1) Under 30 Years									
Original.....	0	Male.....	..	..	..	..	..	..	..
		Female...	..	..	..	..	..	..	..
Literature....	4	Male.....	1	0	0	0	0	0	0
		Female...	5	2	0	2	40	0	40
		Unknown	0	0	0	0	..	..	..
		Total....	6	2	0	2	33	0	33
(2) 30 Years and Over									
Original.....	0	Male.....	..	..	..	..	..	..	..
		Female...	..	..	..	..	..	..	..
Literature....	4	Male.....	8	4	1	5	50	13	63
		Female...	8	6	0	6	75	0	75
		Unknown	1	1	0	1	100	0	100
		Total....	17	11	1	12	65	6	71
4. NEITHER PARENT AFFECTED									
(a) Offspring 30 Years of Age and Over									
Original.....	14	Male.....	20	10	0	10	50	0	50
		Female...	15	8	1	9	53	7	60
Literature....	15	Male.....	26	10	0	10	38	0	38
		Female...	44	17	0	17	39	0	39
		Unknown	14	0	0	0	0	0	0
Total.....	29	Male.....	46	20	0	20	43	0	43
		Female...	59	25	1	26	42	2	44
		Unknown	14	0	0	0	0	0	0
		Total....	119	45	1	46	38	1	39
(b) Offspring Both Over and Under 30 Years of Age									
(1) Under 30 Years									
Original.....	10	Male.....	5	1	0	1	20	0	20
		Female...	12	3	0	3	25	0	25
Literature....	7	Male.....	11	1	0	1	9	0	9
		Female...	6	0	0	0	0	0	0
		Unknown	3	0	0	0	0	0	0
Total.....	17	Male.....	16	2	0	2	13	0	13
		Female...	18	3	0	3	17	0	17
		Unknown	3	0	0	0	0	0	0
		Total....	37	5	0	5	14	0	14
(2) 30 Years and Over									
Original.....	10	Male.....	22	3	4	7	14	18	32
		Female...	23	10	1	11	43	4	48
Literature....	7	Male.....	16	10	0	10	63	0	63
		Female...	18	7	0	7	39	0	39
		Unknown	1	0	0	0	0	0	0
Total.....	17	Male.....	38	13	4	17	34	11	45
		Female...	41	17	1	18	41	2	44
		Unknown	1	0	0	0	0	0	0
		Total....	80	30	5	35	38	6	44

parents affected; (2) mother only affected; (3) father only affected, and (4) neither parent affected. The fraternities tabulated are exclusively those that have at least 1 affected person, and in which some are at least 30 years of age (except in type 1).

In table 5 are given, for the cases taken from the literature, the same classes of data as in table 4.

Table 6 combines in summary fashion the findings of the 2 preceding tables.

4. *Testing of Hypotheses of Inheritance of Otosclerosis.*—Assuming, as is almost necessary in our present state of knowledge, that 1 or more genes are responsible for the peculiar constitution that permits otosclerosis to develop, it remains to determine how many there are and how they are located.

To determine this, various hypotheses were tried out: 1. The hypothesis that otosclerosis depends on a single, dominant autosomal factor, to be tested by mating 2 normal persons (recessives); expectation 100 per cent normal. Hypothesis 1 fails since 2 normal parents (recessives) actually have produced over 80 affected individuals in the families presented.

2. The hypothesis that otosclerosis depends on a single, recessive, autosomal factor, to be tested by mating 2 otosclerotic persons; the expectation is 100 per cent otosclerotic offspring. Hypothesis 2 fails, since 2 otosclerotic parents had only about 70 per cent otosclerotic offspring.

3. The hypothesis that otosclerosis depends on 1 sex-linked dominant factor. This fails, since when neither parent is affected no otosclerosis is expected. Actually in our families 75 cases of otosclerosis occurred.

4. The hypothesis that otosclerosis depends on a sex-linked, recessive gene. This fails since, on this hypothesis, one should find an excess of affected males, as compared with females, as in color blindness. But this is opposed to the facts.

5. The hypothesis that otosclerosis depends on 2 recessive genes, both autosomal. This fails since, according to it, *all* offspring of 2 affected parents should be affected, whereas only 70 per cent are.

6. The hypothesis that otosclerosis depends on 2 recessive factors: x, sex-linked recessive; a, autosomal recessive. This hypothesis demands that when both parents are affected all children shall be. Actually only about 70 per cent are. Also, when the mother only is affected, there is expected an excess of affected sons, but actually sons and daughters are equally affected. Also, when the father only is affected, the proportion of affected children should be small and equal in the sexes.

Actually there is an excess of affected daughters and the proportion of offspring affected is about 43 per cent.

7. The hypothesis that otosclerosis depends on 2 dominant factors, both autosomal. This fails, since the expectation is that the proportion of sons and of daughters affected is equal.

8. The hypothesis that otosclerosis depends on 1 dominant and 1 recessive factor as follows: x, sex-linked recessive; A, autosomal dominant. This fails, since according to it there should be an excess of affected males, but the contrary is the case.

TABLE 7.—*Zygotic and Gametic Formulas for Parents of Otosclerotic Children*

	Female	Male
For otosclerotic parents		
Commonest zygotic type	Xx Aa	YX Aa
Corresponding gametes	XA, xa or Xa, xA	YA, Xa or Ya, XA
Most extreme zygotic condition	XX AA	YX AA
Corresponding gametes	XA	YA, XA
Intermediate zygotic type	Xx AA XX Aa	YX Aa
Corresponding gametes	XA, xA XA, Xa	YA, xa or Ya, XA
For nonotosclerotic parents who have otosclerotic children		
Commonest zygotic type	xx Aa or Xx, aa	Yx Aa
Corresponding gametes	xA, xa or Xa, xa	YA, xa or Ya, xA
Most extreme zygotic type	xx, AA or XX, aa	Yx, AA or YX, aa
Corresponding gametes	xA Xa	YA, xA Ya, Xa

9. The hypothesis that otosclerosis depends on 1 dominant and 1 recessive gene as follows: X, sex-linked dominant; a, autosomal recessive. This fails, as it requires that the daughters of 2 otosclerotic parents shall all be otosclerotic (it is nearly true in Dr. Milles' findings but probably not true generally). Also on this hypothesis an excess of affected females is expected when neither parent is otosclerotic. Actually the proportion of males to females is about the same. This hypothesis is to be kept in mind as possibly good.

10. The hypothesis that otosclerosis depends on 2 factors: X, sex-linked dominant; A, autosomal dominant. This hypothesis may be tested in preliminary fashion, by comparing the summarized expectations (table 7) from this hypothesis with the findings given in table 6.

The facts so far considered suggest the following hypothesis of the genetic basis of otosclerosis: *Otosclerosis develops under external conditions that favor it whenever the patient has a constitution that combines*

2 dominant factors, as follows: a factor X, which lies in the sex chromosome, and a factor, A, which lies in 1 of the autosomes.

5. *Application of Hypothesis that Otosclerosis Requires Two Factors, Both Dominant—One Autosomal, the Other Sex-Linked.*—In applying this hypothesis it will be convenient to make use of a formula such as is common in genetic work. The autosomal factor may be designated by the initial letter "a." "A" then implies the presence of the (dominant) A factor; "a" the recessive condition or absence. "X" indicates the presence of the otosclerotic factor in the sex chromosome; "x," its absence. In the male there is only 1 active sex chromosome; the other, *Y*, we assume to lack any factors, certainly any factor relating to otosclerosis. *Y* is printed in Italics to indicate that in the inheritance of otosclerosis it apparently plays no rôle.

According to this hypothesis a phenotypic otosclerotic female and a phenotypic otosclerotic male have the formulas shown in table 7. The same table gives the formulas for nonotosclerotic parents.

One notes that otosclerotic females have more kinds of zygotic formulas than otosclerotic males have, just because the double X allows a larger number of permutations, viz., XX, Xx, xx. In the male the *Y* is invariable.

Different combinations of these gametes will give differing theoretical proportions of otosclerotic offspring, as shown in table 9. Here the commonest case is that of 2 heterozygotic parents; it is in accord with the usual genetic experience that heterozygotes are commoner than either kind of homozygote. The next commonest case is that of 1 heterozygotic parent.

For example, in group 1, in which both parents are affected, the commonest cases are:

Eggs	Sperm
Xa	Xa
xA	*Y*A
or	or
XA	XA
xa	*Y*a

The female zygotes are as follows: XX, aa (N); XX, aA (o); xX, Aa (o); xX, AA (o); XX, Aa (o); XX, AA (o); xX, aa (N); Xx, Aa (o).[31] This gives 75 per cent otosclerotic and 25 per cent nonotosclerotic offspring. The male zygotes are as follows: X*Y*, aA (o); X*Y*, aa (N); x*Y*, AA (N); x*Y*, Aa (N); X*Y*, AA (o); X*Y*, Aa (o); x*Y*, aA (N); x*Y* aa (N). This gives three-eighths, or 37.5 per cent, otosclerotic offspring and five-eighths, or 62.5 per cent, nonotosclerotic offspring. The remaining lines of table 9 are constructed in similar fashion.

31. (N), normal; (o), otosclerotic.

Thus the order of frequency of the combinations will be about as shown in table 8.

A comparison of the proportions found in fraternities from various matings with hypothesis 10 (table 9) is as follows:

1. Both Parents Affected: This mating is not common. It will ordinarily be true that both affected parents are heterozygous for the 2 dominant genes that are responsible for the defect. In this case 75 per cent of the daughters will be affected and only half of the proportion of sons (37 per cent). However, in 6 of the 8 possible matings of this type, 100 per cent of the daughters are affected and 50 or 100 per cent of the sons. These results also are to be expected from this mating from hypothesis 10. Actually, in Dr. Milles' 3 fraternities all

TABLE 8.—*Types of Gametes of Parents in the Various Matings, Arranged Roughly in Order of Probable Frequency*

	Gametes of Female Parent	Gametes of Male Parent
1. Both parents affected.....	Xa xA or XA xa XA xA XA Xa XA XA	Xa YA or XA Ya XA YA
2. Mother only affected.....	Xa xA or XA xa XA xA XA Xa XA XA	Xa Ya xA Ya or xa YA xA YA
3. Father only affected.....	Xa Xa Xa xa xA xA xA xA xa xa	Xa YA or XA Ya XA YA
4. Neither parent affected, but producing affected offspring	Xa Xa Xa xa xA xA xA xA xa xa	Xa Ya xA Ya or xa YA xA YA

9 daughters were affected (100 per cent)[31a] and 3 of the 5 sons (60 per cent). Thus the proportion of affected sons was three-fifths that of affected daughters. In the cases from the literature the proportion of affected sons and daughters is in each case about 75 per cent, which is given for the second commonest case of this mating. Altogether, the hypothesis of 2 dominant genes is not in disaccord with the proportions found.

2. Mother Only Affected: This mating was fairly common. While in the most probable cases from about 25 to 38 per cent of both daughters and sons are expected to be affected, in the remaining 10 types of mating in this class the proportion of affected offspring is usually higher (from 50 to 100 per cent), and in 2 cases higher in the female offspring than in the male.

31a. In two cases, however, the classification of the hardness of hearing is uncertain.

Actually, in our original cases, among children over 40 years of age the proportion of those who were hard of hearing is 40 per cent for sons and 58 per cent for daughters; in the cases from the literature the ratio is 59:52, and in both series, added together, 54:54. In our original cases and those from the literature the proportion of affected daughters and sons differs somewhat from expectation on the basis of the "commonest" types of matings (being too large a proportion); the sum of all findings shows an approximate equality in the proportion of those affected in both sexes, and this proportion of 45 to 46 per cent is about the average expected in all the types of matings under this heading.

TABLE 9.—*Summary of Proportional Frequency of Otosclerotic and Normal Offspring to Be Expected from Hypothesis 10*

	Females		Males	
	Otosclerotic	Normal	Otosclerotic	Normal
1. Both parents affected				
Commonest case	75	25	37.5	62.5
Next commonest case	75	25	75	25
3 cases	100	0	50	50
3 cases	100	0	100	0
2. Mother only affected				
Commonest case	25	75	25	75
Next commonest case	37.5	63.5	37.5	62.5
4 cases	50	50	50	50
1 case	50	50	25	75
1 case	100	0	50	50
1 case	75	25	75	25
3 cases	100	0	100	0
3. Father only affected				
Commonest case	50	50	0	100
Next commonest case	50	50	25	75
2 cases	50	50	50	50
1 case	75	25	0	100
3 cases	100	0	0	100
2 cases	100	0	100	0
4. Neither parent affected				
1 case	100	0	100	0
3 cases	50	50	50	50
1 case	25	75	25	75
11 cases	0	100	0	100

3. Father Only Affected: This mating differentiates itself from others in the small proportion of affected sons as compared with daughters. In Dr. Milles' cases the sons are just half as frequently affected as the daughters, and when both the original cases and those from the literature are combined, the proportion of otosclerotic sons to otosclerotic daughters is nearly as 2:3. This is the lowest proportion of affected sons found and the greatest difference between the sexes. Thus far the findings are in accord with expectation. That the proportion of affected sons is as high as it is may be due, in part, to the selection exclusively of families with affected children. Numerous families with 1 son, not affected, are excluded from the totals.

4. Neither Parent Affected, but Offspring Affected: The commonest result of the mating of 2 unaffected parents is all unaffected

children. All such matings are excluded from this analysis. The next commonest matings are those in which 50 per cent affected are expected in both daughters and sons. Some matings may give rise to 25 per cent affected children, and matings giving 100 per cent affected are possible. Always, however, an equality of affected sons and daughters is expected.

Actually in our original series the proportion of affected offspring is 50 per cent of the sons to 53 per cent of the daughters; in cases from the literature this ratio is 38:39. To sum up, the proportion of 43 to 42 per cent is found for sons and daughters. This agrees with the expected equal proportion of the 2 sexes affected.

To summarize, the hypothesis of 2 dominant genes is not negatived in any of the groups of matings.

The expected equality of proportion between sons and daughters is realized in group 4. The close approach to equality expected in group 2 is also nearly realized. The excess of affected females expected in group 1 is also found. In group 3 a decidedly small proportion of affected sons as compared with daughters is expected. While the proportion of affected sons actually found seems high, it is relatively lower than the proportion of affected daughters in any other group—as expected.

Since the hypotheses that otosclerosis depends on (1) a single or 2 dominant autosomal genes, (2) a single or 2 recessive autosomal genes, (3) 1 or more sex-linked factors only, (4) a recessive sex-linked and a dominant autosomal gene, (5) 2 recessive genes of which 1 is sex-linked, and (6) a dominant sex-linked and a recessive autosomal gene meet with difficulties in application, the conclusion is reached that the simplest formula which approaches agreement with the findings is that of 2 dominant genes, of which 1 is sex-linked.

In criticism of this general method of discovering truth, it might be urged that so complicated a hypothesis cannot be disproved by any combination of distributions in the offspring. But this conclusion is not justified. On the contrary, as table 9 shows, certain combinations in the offspring are not to be expected when the number of offspring is sufficient to test the case.

Thus, when both parents are affected, some affected children in a fraternity of 2 or more daughters and 3 or more sons are to be expected. They are always found. Again, when the mother only is affected, affected sons will be common. But when the father only is affected, affected sons will be less common. According to table 6, when the mother only is affected, 45 per cent of the otosclerotic offspring of known sex are sons. When the father only is affected, 31 per cent of the otosclerotic offspring of known sex are sons.

(To be Continued)

PROGRESSIVE DEAFNESS OCCURRING IN IDENTICAL TWINS

GEORGE E. SHAMBAUGH, JR., M.D.

WITH A DISCUSSION OF THE FACTOR OF HEREDITY IN THE ETIOLOGY OF DEAFNESS BY

GEORGE E. SHAMBAUGH, M.D.

CHICAGO

In the course of making examinations of some members of the Chicago League for the Hard of Hearing, we came across a pair of identical twins who had become members of the League because they were afflicted with progressive deafness. The situation is such an unusual one that we are reporting our findings.

The twins were women, aged 31, both unmarried, and presented the identical appearance so striking in such twins. There was no history of deafness obtainable in the families of either the father or the mother. In the immediate family there were three sisters, all with normal hearing; one was 35 years of age and one 29, and the third died at the age of 14. The mother was 60 years of age and the father 70. The father had no brothers or sisters, but the mother had six siblings, all living and well, and none was hard of hearing.

Both of the twins had had the usual diseases of childhood, including measles, mumps, scarlet fever and whooping cough; neither had had aural complications or any illness since childhood except for the usual colds in the head.

Deafness appeared in both twins at the same time, when they were 17 years old. The defect came on insidiously without tinnitus aurium. Paracusis was not a symptom in either case. Both had experienced early difficulty in hearing over the telephone, a characteristic of primary nerve deafness. Both had tried using electrical hearing devices, but neither had found much satisfaction with them. Both stated that the hearing seemed worse during attacks of acute colds. The symptom of vertigo had not been present in either case. Both of the twins had had their tonsils and adenoids removed, ostensibly because of the deafness, and, in addition, one had submitted to an operation on the nose, a submucous resection of the septum, to relieve the symptom of obstruction to nasal respiration.

Examination showed the nasal passages to be free from pathologic alterations, and the sinuses on transillumination were clear. The vault of the pharynx appeared free from accumulation of lymphoid tissue. The faucial tonsils had been successfully enucleated in one of the twins, and in the other conspicuous nodules of lymphoid tissue were found at the lower pole of each tonsil fossa. The drum membranes in both women were normal except for an appreciable diminution in luster in each ear. The tubes were readily inflated by the Politzer method.

The functional tests gave identical results in both of the twins, except for slight differences in the degree of deafness. Both gave the functional reactions of primary nerve deafness. In one the Weber test was lateralized, and in the other it

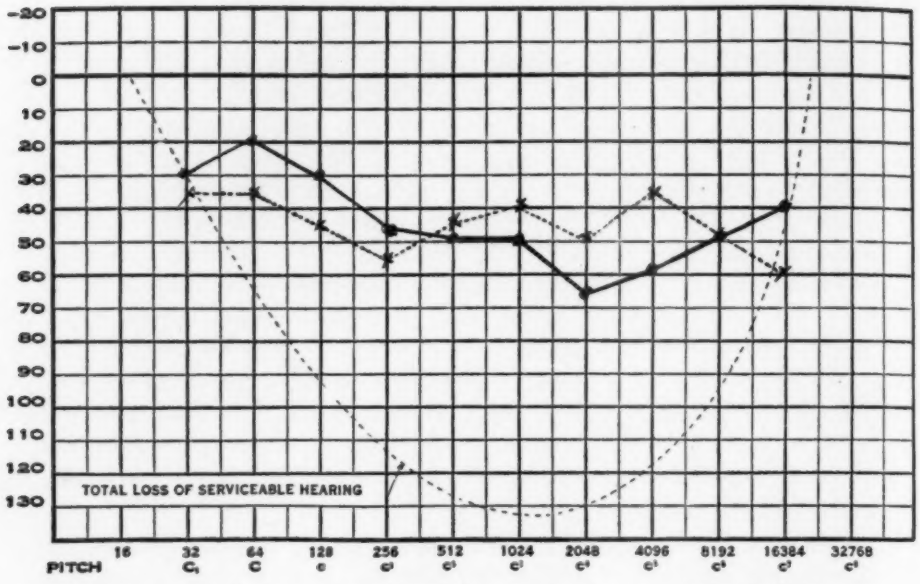

Chart 1.—Audiogram of an identical twin showing the defect in both ears extending throughout the tone range. In this and the following audiograms, the right ear is indicated by the solid line and the left by the broken line.

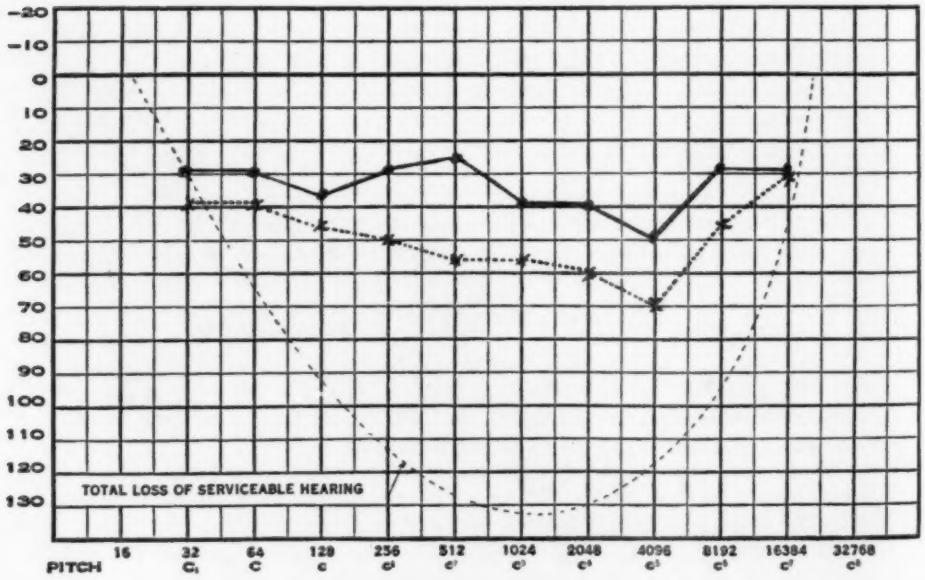

Chart 2.—Audiogram for the other identical twin showing hearing defect in right and left ears throughout the tone range.

was not. Bone conduction with the A Bezold fork was appreciably shortened. The Rinne reaction was positive in both ears for each twin. The hearing for the C_1 fork (32 double vibrations) was slightly shortened in both ears for each twin; the C fork (64 double vibrations) was heard normally in both ears for each, and c^4 (2,048 double vibrations) was moderately shortened in both ears. The whispered voice was heard as follows: In one twin, "76" was heard at 2 feet (60.96 cm.), "82" at 2½ feet (76.2 cm.) in the right ear, and "66" at 1½ feet (45.7 cm.) and "82" at 8 inches (20.3 cm.) in the left ear; in the other twin, "88" and "66" were heard close to the right ear and at 2 inches (5 cm.) from the left ear.

In testing the vestibular mechanism, the nystagmus in one of the twins lasted twenty seconds after rotation to the right as well as to the left; in the other, the nystagmus after rotation to the right lasted fifteen seconds and after rotation to the left sixteen seconds.

Audiograms made with a 1-A Western Electric audiometer showed a defect throughout the tone range with only minor differences for the two women (charts 1 and 2).

HEREDITY IN THE ETIOLOGY OF DEAFNESS

The conclusion from these findings is that both of the twins are suffering from primary nerve deafness. In both the condition presented is that of progressive loss of hearing. The fact that the process began insidiously in the second decade of life leads one to suspect that the underlying pathologic process may be otosclerosis in which the changes in the capsule have not encroached on the foot plate of the stapes. That heredity is the important etiologic factor is apparent, since in both persons the process came on simultaneously.

Macfarlan,[1] in his article on "Identical Hearing in Identical Twins," published audiograms showing the hearing defect in identical twins. The subjects were women in middle life. No statement is published regarding the probable duration of the defect or any other functional tests which might determine whether the alteration in hearing was the result of obstruction to sound conduction or was due to degeneration of the nerve itself.

Judging alone from the audiograms which were published, there was a defect throughout the tone range with a depression between 256 and 2,048 double vibrations and a sharp falling off above 4,096 double vibrations. The audiograms for the two women were almost similar and conform quite accurately to the type observed so frequently in cases of primary fixation of the stapes with a defect in the upper tone ranges.

From the meager statement included in this report, one can only say that the hearing defects were similar in the two ears and in the two women. One may surmise that these were cases of otosclerosis, in which heredity is known to play such a conspicuous rôle (chart 3).

The importance of heredity in profound deafness in childhood was brought out in the examination of 5,348 children in our public institu-

1. Macfarlan, Douglas: Laryngoscope **37**:846 (Nov.) 1927.

tions for the deaf, which was carried out under the auspices of the National Research Council.[2] These studies revealed that 60 per cent of profound deafness in childhood is congenital and 40 per cent acquired. Heredity and consanguinity were important factors in the causation of these cases of congenital deafness, a family history having been present in one third of the cases and consanguinity in 7 per cent. In 112 instances there were deaf uncles, in 126 instances deaf aunts and in 175 instances deaf cousins. In 144 cases one or both parents were deaf, in 40 cases grandparents and in 59 cases other relatives. The figures showing deafness in the immediate family, that is, the number of other children deaf, were of particular interest. In 484 cases another child

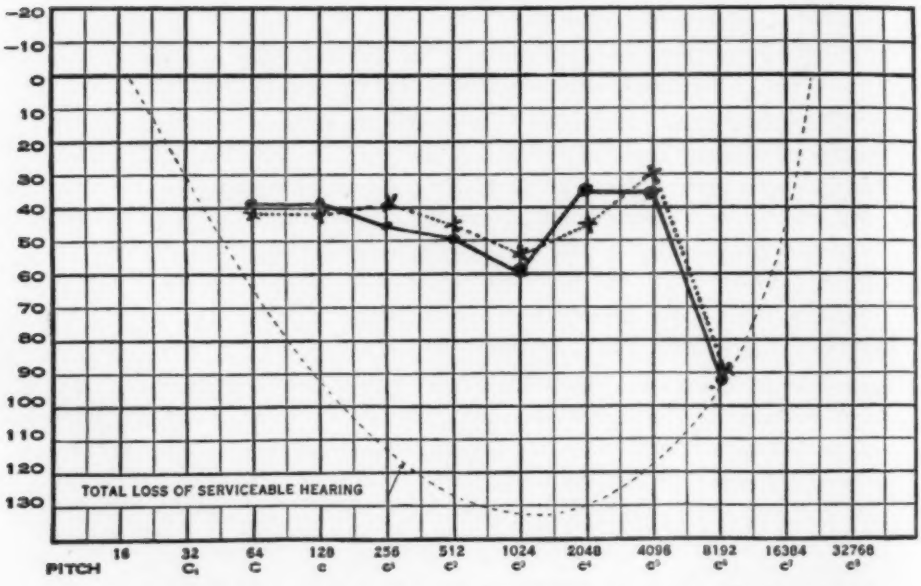

Chart 3.—Audiogram made by Dr. Macfarlan of one of a pair of identical twins showing the defect in the right and left ears throughout the tone range, with the depression at 1,024 double vibrations and a sharp drop at 4,096 double vibrations. The audiogram for the other identical twin reported by Dr. Macfarlan is almost the same.

in the same family was deaf, in 211 two others, in 47 three others, in 13 four others and in 6 five others; 17 pairs of twins were deaf.

The 40 per cent afflicted with acquired deafness were found to have lost their hearing, for the most part, through the toxic action of infection on the nerve of hearing. Otitis media, acute and chronic, was

2. Shambaugh, G. E.; Hayden, D. B.; Hagens, E. W., and Watkins, R. W.: Statistical Studies of the Children in the Public Schools for the Deaf, Arch. Otolaryng. **12**:190 (July) 1930.

found responsible for profound deafness in only a small number of these children. Heredity, of course, plays no part in these cases of acquired deafness.

Aside from the children afflicted with profound deafness and cared for in our public institutions for the deaf, there are a great number of children of school age who are known to be afflicted with mild defects in hearing. These defects appear, for the most part, to be the results of tubotympanic processes which vary from a mild, simple, catarrhal otitis media to a suppurative disease. In this type of deafness heredity plays no part except in so far as there may be a hereditary tendency for the development of lymphoid hypertrophies in the nasopharynx.

What becomes of these cases of tubotympanic disease so common in childhood is well understood. In very few does the process continue active after the age of puberty, and in scarcely any does the middle ear condition produce marked defects in hearing. It is only in the exceptional case that the hearing defect continues to progress in adult life and, in at least some of these, the progress results from a recurrence of acute exacerbations of the middle ear process. More often the cause of the progressive deafness in adult life which develops in persons who were in childhood afflicted with tubotympanic processes is otosclerosis.

The many adults with severe deafness consist, for the most part, of persons who have acquired their deafness after the age of puberty as a result usually of progressive deafness which began to show itself in adult life. Progressive deafness in these cases, appears, for the most part, to be the result of otosclerosis, a disease involving the bony capsule of the labyrinth, which produces in most cases fixation of the stapes and, with this fixation, the typical functional reactions of obstruction to sound conduction with the defect in the lower tone ranges particularly marked.

That otosclerosis may develop without fixation of the stapes and with the clinical findings characteristic of primary nerve deafness is amply demonstrated both from clinical observation and through pathologic findings. Postmortem examinations, moreover, have shown that otosclerotic changes in the bony capsule are present in cases in which the process had not been suspected during life.

Heredity appears to be the most important factor in the etiology of otosclerosis. This does not mean that every person in whom this clinical diagnosis can be made gives a familial history of otosclerosis. Hereditary processes of whatever sort often remain recessive in a person, but will be transmitted to the offspring.

In the prevention of otosclerosis the hereditary factor is the most important one which can be utilized. Persons in whom there is a history of familial deafness should be urged to refrain from bearing children. The fact that heredity is the only etiologic factor known at

present does not mean that the matter should rest here. One is still hopeful that activating causes which stimulate development of a latent otosclerosis may be detected and the process prevented in spite of an hereditary factor.

In conclusion, I would point out that heredity appears to be the most important factor in the etiology of profound deafness, whether this occurs in children or is the result of otosclerosis in adult life. The study of otosclerosis in identical twins may help to emphasize the relative importance of heredity in these cases, and perhaps some light on other etiologic factors may be disclosed, for no two persons pass through life with identical experiences, although in identical twins the hereditary factor is exactly the same. If otosclerosis is observed to develop in one of a pair of identical twins and not in the other, it seems probable that a careful search into the experiences of the two persons may bring to light facts which will point toward an activating cause for this obscure disease. If, on the other hand, the occurrence of otosclerosis in identical twins is always the same no matter what the individual experiences of the two may be, this fact would indicate that heredity is the all-important factor in the etiology.

ADDENDUM

Since submitting this manuscript for publication, we have had the opportunity of examining another pair of identical twins afflicted with progressive deafness.

The twins were men 69 years of age. The history of deafness in the family was as follows: The father died at the age of 58, when he was known to be slightly deafened; the onset of his deafness was not known. A brother of the father became quite deaf in old age; the onset was stated to be around 80, and the defect was insidious and progressive. A son of this uncle became quite deaf in old age, the defect being progressive and coming on insidiously; it was first recognized at the age of 60. On the mother's side there was no deafness.

The history of the deafness in the twins was as follows: The onset was insidious, coming on about the same time in both; it was first recognized after the age of 60 and may have begun a year or two earlier in one than in the other. The defect in both men was bilateral and progressive. There was no history of any previous ear trouble even in childhood. An occasional slight tinnitus was noticed in but one and had been present only for the last three years. Paracusis was not present in either. Both found especial difficulty in hearing over the telephone, and both had tried electrical hearing devices but found them of little assistance; this is characteristic of primary nerve deafness. The state of the general health of both men was good. Neither had suffered from any serious illness since childhood. One had had no local treatment for the deafness; the other had had a few inflations without relief.

The nasal passages were normal in both men. By transillumination, the frontal and maxilliary sinuses of both were clear. The tonsils in both were small and presented the usual evidence of chronic tonsillitis at this age of life. In one of the twins the right and left drum membranes were normal, but in the other they showed

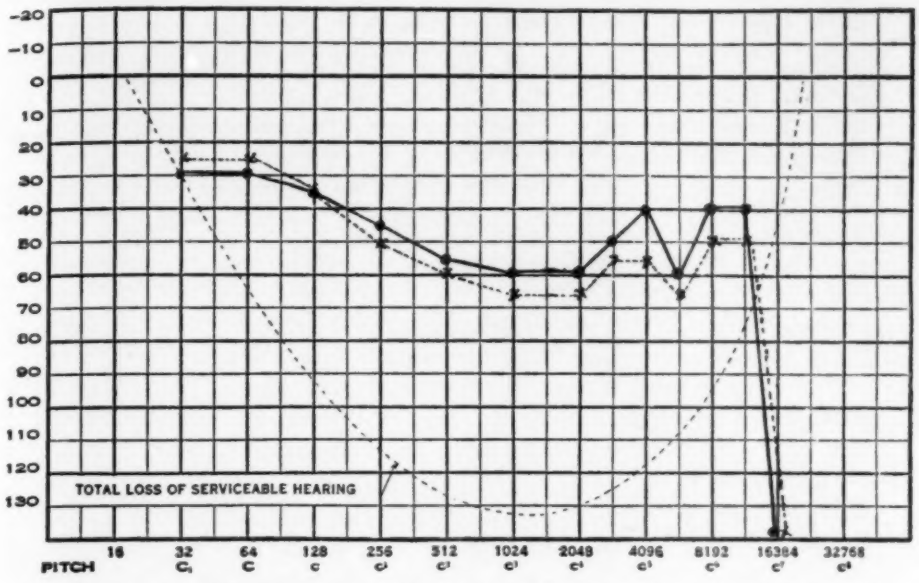

Chart 4.—Audiogram of an identical twin with progressive deafness, the onset of which occurred late in life.

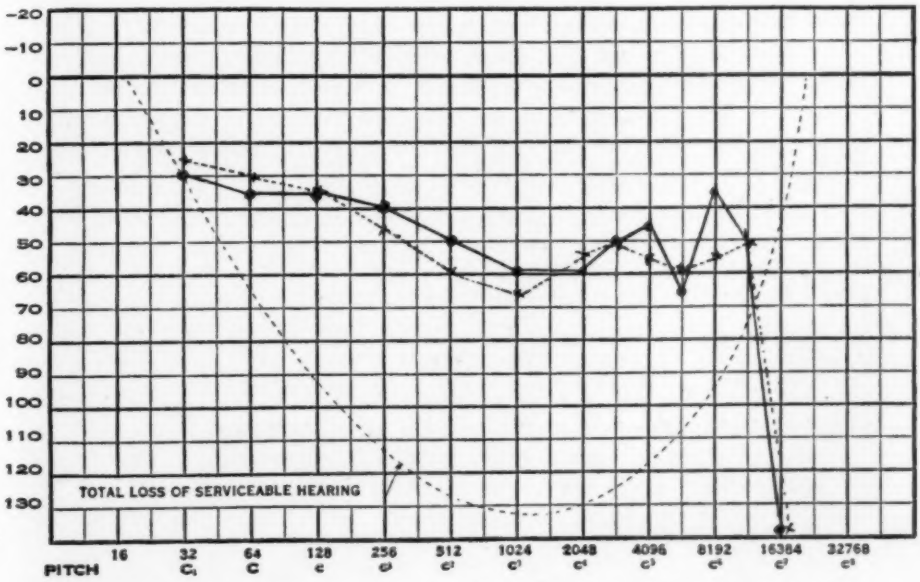

Chart 5.—Audiogram of the other identical twin with progressive deafness.

some thickening and diminution of luster. In both, the tubes were readily inflated by the Politzer method, but without any effect on the hearing.

The functional hearing tests were as follows: In one of the twins the whispered voice was heard close to the ears; in the other, it was heard close to one ear and 4 feet from the other ear. The Weber test was lateralized in one to the right and in the other to the left, the A fork being used. The Schwabach test, with the A fork, was distinctly shortened in both men. The C_1 (32 double vibrations) was slightly shortened for one in both ears, and scarcely heard by the other in both ears. C (64 double vibrations) was normal for both of the twins; c^4 (2,048 double vibrations) was very much shortened and the Rinne test was positive in both ears for each twin. The vestibular responses after rotation were entirely normal. The audiograms presented practically identical curves for both ears for each twin.

Comment.—The defect in both of the twins is clearly one of primary nerve deafness, which, coming on insidiously after the age of 60, would ordinarily be passed on as due to senile degeneration. The fact that there was a history of familial deafness on the father's side, coming on also late in life, could be interpreted as due to the same etiology. Having observed similar instances of progressive nerve deafness coming on after 50, leading to severe deafness, when other immediate members of the family were known to be afflicted with otosclerosis, we are led to question the possibility of some of these cases being the labyrinthine type of otosclerosis.

The characteristic audiogram in senile deafness shows a sharp depression as one approaches the higher tone range with relative preservation for the middle and low tones, whereas the audiograms of these two show a depression most marked in the middle tone range with comparatively good hearing for the higher ranges, found so frequently in otosclerosis.

In this issue of the ARCHIVES (p. 179) appears a report by Dr. Frank A. Rodin on the hearing defect in a pair of identical twins in whom the clinical diagnosis of otosclerosis with primary fixation of the stapes was made. This makes altogether four instances of progressive deafness occurring in identical twins. In all four, both ears were similarly and simultaneously involved. Of these four instances in identical twins, one (Dr. Rodin's cases) was definitely otosclerosis with primary stapes fixation; one (Dr. Macfarlan's cases), judging from the data, might have been otosclerosis, while the remaining two instances which we report were possible cases of otosclerosis without stapes fixation. The fact that in all four instances the defect in hearing was approximately equal in the twins and came on at nearly the same time suggests that in otosclerosis heredity is the all-important etiologic factor.

IDENTICAL HEARING DEFECT IN IDENTICAL TWINS

FRANK H. RODIN, M.D.
SAN FRANCISCO

The resemblance of identical twins to each other in mental characteristics, dispositions, likes and dislikes is well recognized. The close resemblance of their features, such as birth marks, finger prints, sole prints and blood groups, has been studied and reported. The study of resemblances of identical twins is of great importance, as it throws some light on the inborn factors in health and in disease.

The only report of a hearing defect in identical twins with audiogram findings that I have been able to find is that of Macfarlan,[1] who reported the cases of middle-aged twin sisters who were teachers. These twins were identical in every respect: They looked alike, were of the same height and weight, acted alike, thought alike and dressed alike. Their audiograms showed the same loss of hearing: The falls and rises across the pitch range were in nearly exact correspondence one with the other. Neither of the twins had had any previous otitic trouble.

REPORT OF CASE

History.—Misses Lillian and Violette V., aged 15, who were found to have defective hearing when tested with the phonograph audiometer,[2] were tested with the 2-A Western Electric audiometer and were found to have practically identical hearing defects, as shown by the audiograms.

The parents were living. There was one sister, who was alive and well. There was no history of any deafness or hardness of hearing in the family.

At birth the patients were frail, and they grew slowly. The only illnesses that they had had were scarlet fever and whooping cough, which both had at the same time. According to the mother, when the sisters were 6 years of age they had scarlet fever. Following this the hearing began to decrease gradually until about three years prior to examination, when it became stationary and had not changed since then. The sisters had known for some time that their hearing had been poor, and according to them it had not changed for many years. At no time did they have any disease of the ears.

Examination.—This showed two healthy girls identical in every respect. Their weight was the same, 93 pounds (42.2 Kg.). Lillian was 59 9/10 inches (150.75 cm.)

From the Bureau of Child Hygiene, San Francisco Department of Public Health; J. C. Geiger, M.D., Director of Public Health.

1. Macfarlan, D.: Identical Hearing in Identical Twins, Laryngoscope **37**:846 (Nov.) 1927.

2. Rodin, F. H.: Survey of the Hearing of the School Children of San Francisco, Arch. Otolaryng. **11**:463 (April) 1930.

tall, while Violette was 58⅞ inches (149.05 cm.) in height. Their hair was brown, Lillian's hair being perhaps slightly lighter. Their eyes were the same color, blue.

The patients had no symptoms of vertigo or paracusis willisiana, and had no great difficulty in using the telephone. They gave an identical complaint of tinnitus aurium, and they described it as an intermittent ring in both ears, like a bee buzzing, the same in each ear.

Examination was conducted by Dr. Irvine, and revealed the following: The tonsils were present, with some lymphoid tissue on the posterior pharyngeal wall.

Results Obtained for Various Tests on the Ears of Lillian and Violette and Comparison with the Normal *

Test	Normal	Lillian		Violette	
		Right	Left	Right	Left
Spoken voice, heard at	30 ft.	12 ft.	12 ft.	15 ft.	15 ft.
Whispered voice, heard at					
For numbers like 66	16 ft.	0	1 ft.	0	1 ft.
For numbers like 18	12 ft.	0	0	0	0
Ingersoll watch	36 in.	Not heard		Not heard	
Tuning forks, length of time heard in seconds					
24 double vibrations	4	0	0	0	0
48 double vibrations	4	0	0	0	0
64 double vibrations	45	4	4	4	4
128 double vibrations	30	10	5	8	11
256 double vibrations	60	8	7	8	12
512 double vibrations	50	13	24	8	18
1,024 double vibrations	50	12	14	10	8
2,048 double vibrations	25	8	3	17	13
Galton whistle	..	Definitely heard by either ear			
Weber's	..	Not localized		Not localized	
Test with A tuning fork					
Air conduction	90	55	55	55	55
Bone conduction	60	60	60	60	60
Rinne's	..	Negative	Negative	Negative	Negative
Tinnitus aurium	..	Intermittent ring in both ears; like a bee buzzing, but not localized			
Paracusis willisiana	..	Not present		Not present	
Vertigo	..	Not present		Not present	
Rotating	..	Normal reaction		Reaction slightly diminished	
Caloric	..	Not performed		Reaction slightly diminished	

* The tests were conducted by Dr. R. S. Irvine. The normal used in this table is the normal for Dr. Irvine's tuning forks.

The same type of nasopharynx was seen in each case, except that Lillian had some postnasal discharge. Each sister showed the same nasal findings: The septum was moderately deflected to the left; there were no other abnormalities. There was a slight retraction of the tympanic membrane, a little less in the case of Lillian, but within normal limits.

Tests of Hearing.—Lillian heard the spoken voice at 12 feet (365.76 cm.); Violette at 15 feet (457.2 cm.), as shown in the accompanying table. The whispered voice was not heard in the right ear; with the left ear, each sister heard numbers like 66 at 1 foot (30.48 cm.), but did not hear numbers like 18. The Ingersoll watch was not heard. Tuning forks with 24 and 48 double vibrations were not heard. The other tuning forks were heard, but the time duration heard was greatly shortened (chart 1). A Galton whistle was heard distinctly by either

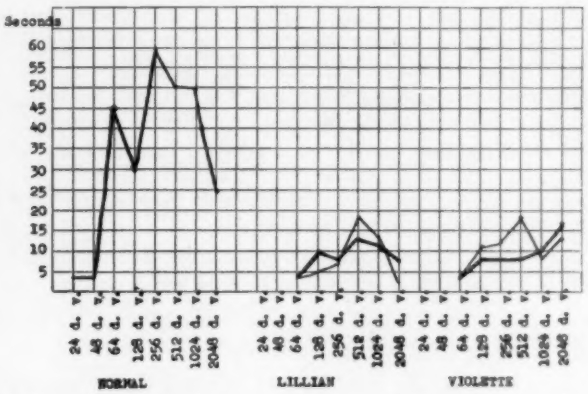

Chart 1.—Curves showing the normal response to the tuning forks and that obtained from Lillian and Violette. The heavy black curve is for the right ear; the lighter black curve, for the left ear.

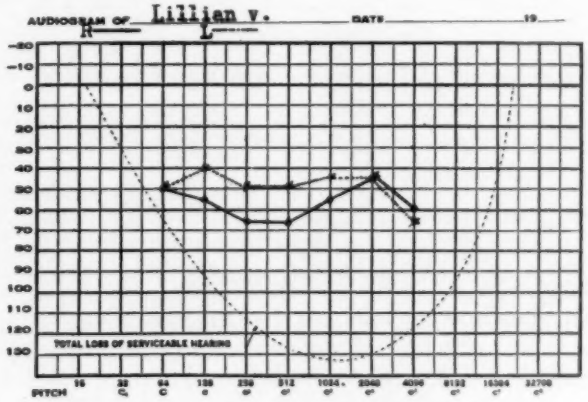

Chart 2.—The audiogram of Lillian's hearing.

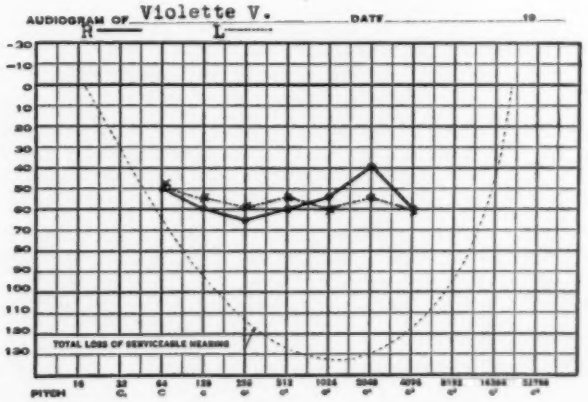

Chart 3.—The audiogram of Violette's hearing.

ear. Weber's test was not localized. In testing with the A tuning fork, air conduction was found to be greatly diminished, but bone conduction was normal. Rinne's test gave negative results. The rotating test showed a normal vestibular reaction with Lillian, but a slightly diminished reaction with Violette. The caloric test was done for Violette, and this also showed a slightly diminished reaction.

The mental reaction time to all the tests was slow, and the sisters became exhausted easily.

The audiograms (charts 2 and 3) showed that both sisters had an identical loss of hearing in each ear for tone of 64 double vibrations. For the remaining pitch range the loss of hearing was nearly the same with each sister, while for a tone of 4,096 double vibrations the loss of hearing was the same in Lillian's right ear and in Violette's right and left ears. Neither Lillian nor Violette had any response to a tone of 8,192 double vibrations.

Otosclerosis is probably the correct clinical diagnosis, as there was an insidious type of conductive deafness in two young girls without any apparent cause, with low-pitched tinnitus aurium, a negative Rinne test, a normal Schwabach test and normal tympanic membranes.

SUMMARY

1. The case reported is that of two sisters, identical twins, with identical loss of hearing.

2. Functional hearing tests showed practically the same loss of hearing for air conduction.

3. Weber's test was not localized, and Rinne's test gave negative results with each sister.

4. The audiograms are practically identical.

5. The diagnosis of otosclerosis was made in view of the insidious type of deafness without any apparent cause in two young girls, a negative Rinne test and normal tympanic membranes.

490 Post Street.

PYEMIA FOLLOWING ACUTE TONSILLAR INFECTIONS

HERMAN RUBIN, M.D.
BROOKLYN

Pyemia following acute tonsillar infections is not a rare occurrence, although only a few cases have been reported in the American literature. This is probably because this condition is not recognized, or is recognized too late. Frequently surgical procedures are carried out after the infection has extended beyond the venous system of the head and the neck.

According to Nussbaum,[1] only fifty-eight cases of posttonsillar pyemia had been reported in the literature prior to 1927. In this group there were thirteen recoveries following ligation and excision of the jugular vein.

REPORT OF CASES

CASE 1.—C. W., aged 24, a plumber, a patient of Dr. Myerson, gave a history of severe sore throat, pain on swallowing, slight hoarseness and some aching in the limbs of four days' duration. His past history was irrelevant. Examination revealed an extremely ill young man. The tonsils were enlarged and covered with a thin grayish-white exudate. The remainder of the throat was moderately red. There were edema of the uvula and slight edema of the upper pole of the left tonsil and the mucous membrane immediately surrounding it. The temperature at the time of the patient's admission was 103.2 F.; the pulse rate was 100; the respiratory rate was 20. A diagnosis of streptococcic tonsillitis was made, and 5,000 units of strepto-erysipelas antitoxin was administered. The patient had several chills, and his temperature ranged between 98 and 105 F. during the next six days. On the third day, a slight jaundice appeared. Some swelling over the left side of the face was noted at the time. It was apparent that the patient was suffering from a septicemia which was attributed to the preexisting tonsillar infection.

The laboratory findings were as follows: red blood cells, 5,220,000; hemoglobin, 90 per cent; the white blood cells ranged between 15,400 and 19,400, with from 82 to 86 per cent polymorphonuclears. Within the next few days, the hemoglobin dropped from 90 to 76 per cent and then to 60 per cent. A culture of the tonsillar region yielded Staphylococcus albus. The urine contained bile and a slight trace of albumin. Blood cultures on four successive days were sterile.

An injection of 100 cc. of 1:5,000 acriflavine intravenously and a transfusion of 250 cc. of blood were given. On the sixth day operation was performed under local anesthesia. When the carotid sheath was exposed, a considerable matting of

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From the Department of Otolaryngology, Kings County Hospital, Service of Dr. M. C. Myerson.

1. Nussbaum, D.: Pyemia Following the Anginas, Laryngoscope **39**:787 (Dec.) 1929.

glands about its upper portion was found. The internal jugular vein was thickened and discolored. This was ligated above and below the facial vein. Pus was not encountered in the neck.

The temperature rose to 107 F. the day after operation. The patient vomited a small amount of bile-stained material and was delirious. On the second day after operation he had a prolonged chill. His temperature rose to 108 F.; he became comatose and died.

Autopsy revealed gangrenous changes in the deep structures of the tonsillar tissue, thrombophlebitis of the left internal jugular vein, multiple pulmonary abscesses, empyema of the right pleural cavity and cloudy swelling of the liver, spleen and kidney.

Comment.—This patient should have had earlier surgical intervention, but I was handicapped by my inexperience. Swelling was not present along the sternocleidomastoid muscle in this case. Unfortunately, an examination of the tonsillar and the facial veins was not made.

Case 2.—A carpenter, aged 33, was admitted to the hospital with a history of a sore throat for five days accompanied by chills, fever and sweats. He had a chill on the second day of his illness and two chills on the fourth day. Examination of the throat showed both tonsils to be enlarged and acutely inflamed. There was a grayish-white exudate on the left tonsil. There was some edema of the upper pole and of the mucosa around this area. On the next day there was swelling of the left side of the face, as well as swelling behind the angle of the jaw, which was due to enlarged upper cervical glands. While the patient was in the hospital he had several chills followed by a rise in temperature to 106 F. Ten cubic centimeters of strepto-erysipelas serum was given intramuscularly. The blood count on admission showed 13,600 white cells, with 90 per cent polymorphonuclears. On the day after admission, a diagnosis of pyemia following acute tonsillitis was made, and operation was performed to expose the internal jugular vein on the left side.

Considerable difficulty was encountered in exposing the carotid sheath because of numerous glands in the jugular triangle. The vein was found thickened and somewhat discolored. A double ligature was placed above the facial vein. No pus was found in the neck. The temperature ranged from 99.3 to 106 F. during the four days following the operation. The patient became jaundiced on the first day after operation, and vomited a small amount of bile-stained material. He had several chills during the next few days.

The white blood cells averaged 30,000 after operation, the polymorphonuclear cells averaging about 85 per cent. The hemoglobin did not go below 82 per cent. The red blood cells numbered 4,256,000. Culture from the tonsillar area revealed streptococci, staphylococci and a gram-negative bacillus. The blood culture was negative on two successive days. Three days after operation the patient became extremely toxic, the jaundice increased, and the abdomen became distended. Cheyne-Stokes breathing supervened, and the patient died the next day with what appeared to be terminal bronchopneumonia. During his illness several transfusions of whole blood were given.

Autopsy disclosed: thrombophlebitis of the left lateral sinus, localized meningitis in the region of the temporal lobe, empyema of the right pleural cavity, multiple septic infarcts of both lungs, acute nephritis and cloudy swelling of the myocardium.

Comment.—This patient died of a fulminating infection at the end of ten days. There was no swelling in the neck in this case, nor was

any pus found in the neck. A clot was found in the lateral sinus, but none was present in the jugular vein. The tonsillar and facial veins were not examined.

CASE 3.—J. S., aged 60, a laborer, was admitted with a history that he had had a soreness of the left side of his throat for the past two days. The following day he noticed pain and swelling in the left side of his neck. He had a chill with fever and sweat. His previous history indicated that he had had an operation performed on the right side of his neck for Ludwig's angina, one year before. He had had a moderate discharge from his right ear for the past forty years.

The temperature at the time of admission was 105.2 F.; the pulse rate was 100, and the respiratory rate was 20. Examination revealed an extremely sick man with a temperature range of from 102 to 106 F. There was a necrotic grayish-white membrane on the left tonsil. A swollen gland was present at the angle of the left jaw. Some edema of the upper tonsillar and peritonsillar area was noted. A moderate amount of swelling was present along the course of the anterior margin of the left sternocleidomastoid muscle, with some tenderness on deep pressure, especially about the junction of its middle and lower third. The canal of the right ear was covered with polypoid granulations, so that it was impossible to tell whether any portion of the drum or ossicles was present. There was a small amount of discharge in the canal, with a slight odor. There was no physical or clinical evidence of acute disease of the mastoid.

The left ear was normal. Roentgen study of the right mastoid showed a sclerotic condition, with a complete absence of all cell structure. A roentgenogram of the chest showed no pleural or pulmonary pathologic changes.

The blood count showed: red cells, 4,228,000; hemoglobin, 85 per cent; white cells, 7,500, with polymorphonuclears, 64 per cent. The spinal fluid was clear and showed a pressure of 4 mm. of mercury. Pressure over the right jugular vein caused this pressure to rise to 11 mm., while on the left side the rise was to 5 mm., which indicated a probable block on the left side. Blood culture yielded a hemolytic streptococcus. A diagnosis of pyemia caused by thrombophlebitis of the left internal jugular vein due to streptococcic tonsillitis was made.

The patient had several chills. His temperature ranged between 100.3 and 105.3 F. Operation on the left side of his neck was advised, but permission was withheld until the fifth day. The blood culture was again positive for the hemolytic streptococcus; the chemical composition of the blood was within normal limits, and the spinal fluid was normal.

Operation was performed under local anesthesia. An abscess containing about ½ oz. (15 cc.) of pus was encountered along the middle portion of the carotid sheath. The carotid sheath was opened. The jugular vein was found to be firm and hard, and was thrombosed from the facial vein downward. The facial, lingual and superior thyroid veins were tied off; the jugular vein was resected down to the clavicle. The suture on the clavicular end slipped off, but no bleeding occurred, demonstrating that the thrombus extended into the innominate vein.

For three days following operation the patient seemed to be doing well. On the fourth day after operation he died. An autopsy was not obtained.

Dr. Hala reported that the jugular vein was the seat of an acute septic thrombophlebitis.

Comment.—Because of the suppurating ear on the right side, the clinical picture might have been due to a thrombophlebitis of the internal jugular vein on either side. This patient probabbly had an extensive

thrombosis which extended downward into the subclavian or innominate vein and upward into the bulb and lateral sinus.

CASE 4.—R. K., aged 24, a baker, was admitted to the hospital on April 29, 1931, with a history of sore throat of ten days' duration. The temperature on admission was 104 F.; the pulse rate was 110, and the respiratory rate was 24. Examination revealed an acute follicular tonsillitis. Three days later a retrotonsillar abscess developed on the left side, which was incised, and about 1 drachm (3.75 cc.) of pus was obtained.

Seven days later a swelling developed along the anterior border of the sternocleidomastoid muscle, which was painful and tender on pressure. The temperature at this time was 102 F.; the pulse rate, 100, and the respiratory rate, 24. The blood count showed: red cells, 4,224,000; hemoglobin, 85 per cent; white cells, 17,900, with 77 per cent polymorphonuclears. There were no chills at this time.

Under avertin anesthesia, an incision was made along the anterior border of the sternocleidomastoid muscle, from the mastoid process to the clavicle. The muscle was retracted and the carotid sheath exposed. An abscess was found along the upper portion of the carotid sheath on its outer side, containing about 1 ounce (30 cc.) of pus. The mediastinum was protected with strips of iodoform gauze. The carotid sheath was not opened.

Examination of the pus showed a gram-positive coccus in short chains. Two days later the blood culture was reported positive for a nonhemolytic streptococcus. One week later an abscess developed in the left gluteal region, which was incised. Three days after this the right wrist became red, painful and tender, which condition subsided under local treatment. While the patient was in the hospital his temperature ranged from 98 to 103 F. for a period of forty days. He was discharged well on the forty-fifth day.

Comment.—This patient had a retrotonsillar abscess followed by an abscess in the parapharyngeal space, with pyemic manifestations in the gluteal region and wrist. He recovered without ligation of the internal jugular vein.

REVIEW OF THE LITERATURE

Numerous reports of cases of pyemia following acute tonsillitis are to be found in the German literature. There appear to be two different views as to the method of production of the infection of the blood stream. One view maintains that infection is carried into the blood by means of a suppurating gland or parapharyngeal abscess which is in close proximity to the jugular vein. The other upholds the theory that the small tonsillar veins carry the infection, which frequently attacks the jugular vein by way of a thrombophlebitic process. In some cases, however, the infection travels only by way of the small local blood vessels.

Fränkel,[2] as a result of autopsies in fifteen such cases, found that pyemia originating in the lymph nodes is rare. Chills may be absent, while tenderness of the side of the neck is frequent. He found that

2. Fränkel, E.: Pyämie nach Angina, Deutsche med. Wchnschr. **52**:93 (Jan. 15) 1926.

the process usually begins by direct infection of the tonsillar veins. Anaerobic bacteria were most frequently found. Streptococci and pneumococci were found in several cases. Ligation was done in six of his cases, and all of the patients died.

Melchior [3] ligated the jugular vein in two of his patients with thrombophlebitis and sepsis after acute tonsillitis; both patients recovered. A thrombophlebitic process was discovered in the veins of the tonsils, which extended into the jugular vein.

An interesting case was cited by Eagleton.[4] He described a case of acute otitis with mastoiditis on the right side. At autopsy thrombophlebitis of the left internal jugular vein and lateral sinus was found. He believed that this condition was secondary to acute tonsillitis. This case somewhat resembles case 3.

Uffenorde [5] maintained that the operative and postmortem findings in these cases can be better explained by the spread of infection along the lymph channels to the internal jugular vein and other veins outside the immediate region of the tonsillar focus. He advised that the internal jugular vein should be ligated only if the presence of thrombosis is demonstrated, and in these cases the thrombosed portion should also be resected. If there is peritonsillar inflammation, the approach to the primary focus should be from the outside of the neck. If the jugular vein is not involved but it is evident that the infection has its source in the tonsils or is possibly extending upward, the tonsils can be reached from below and behind the angle of the jaw; at the same time the parapharyngeal space is exposed. This is indicated whether there is an actual abscess or only a phlegmonous process in this region. Uffenorde does not remove the tonsil until later in such cases.

In a later paper, Uffenorde [6] reported three cases of pyemia following tonsillectomy and adenoidectomy. Suppuration was found in the retropharyngeal and parapharyngeal tissues in all. The suppurative lesions were drained, and the veins of the neck were exposed, but no evidence of phlebitis was found in the jugular vein or other veins.

Stahl [7] reported a case in which a tonsillar abscess formed and ruptured spontaneously on the right side. The left side became painful,

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3. Melchior, E.: Pyämie nach Angina, Zentralbl. f. Chir. **52**:1993 (Sept. 5) 1925.

4. Eagelton, W. P.: Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses, New York, the Macmillan Company, 1926, p. 83.

5. Uffenorde, W.: Ueber postanginöse Pyämie, Deutsche med. Wchnschr. **52**: 1638 (Sept. 24) 1926.

6. Uffenorde, W.: Weitere Beiträge zu der von den Mandeln ausgehenden Sepsis, Deutsche med. Wchnschr. **55**:775 (May 10) 1929.

7. Stahl, R.: Zur Kenntnis der Thrombophlebitis und Sepsis postanginosa, Deutsche med. Wchnschr. **53**:186 (Jan. 28) 1927.

and within a few days chills developed. One week after the onset of the illness the patient had several chills daily and a septic temperature. Signs of bronchopneumonia at the base of each lung developed, but disappeared within a few days. No sign of thrombosis of the jugular vein could be demonstrated. A gland on the left side was slightly swollen and tender. A perineal abscess developed and opened spontaneously. Various drugs, as well as a transfusion of immune blood, were used to combat the sepsis, without avail. Operation was undertaken three weeks after the patient's admission to the hospital. The left internal jugular vein was ligated and resected. The ligature passed through the thrombus, which extended beyond it, possibly to the subclavian vein. The patient died on the thirty-ninth day.

At autopsy, multiple abscesses of the lungs, myocardial degeneration, hemorrhagic nephritis, cloudy swelling in the liver and slight hemorrhages in the gastric mucosa were found. This case and others reported in the literature demonstrate that thrombosis may be present without a cordlike swelling or even tenderness along the course of the jugular vein. Stahl therefore urged that if chills and fever develop after local improvement or apparent healing of angina, the internal jugular vein should be ligated.

Claus [8] has reported numerous cases of sepsis following sore throat in several communications. He has done a great deal of work in this field. He [9] called attention to the fact that the diagnosis is difficult in those cases in which the angina has subsided, because the tonsils and pharynx show no sign of inflammation. Even in the most severe cases a cordlike swelling along the course of the jugular vein may not be present. On the other hand, there is always a tenderness to pressure at the angle of the jaw. The history is of special importance. The angina may have occurred as long as four weeks previously. Anaerobic organisms are usually found. In all cases a perivascular infiltration has been present in the peritonsillar connective tissue, and from this infiltration the veins have become involved—sometimes the tonsillar veins, sometimes the facial vein and, at other times, the jugular vein. Claus advocated exposure and drainage of the connective tissue spaces around the tonsils. If the blood vessels show pathologic changes, they should be ligated and excised. If there is a palpable external swelling in the neck, prophylactic mediastinotomy should be performed. He has had recoveries in cases in which the thrombosis has extended from the innominate or subclavian vein to the torcula. This author always per-

8. Claus, H.: Bemerkungen zur Pyämie nach Angina, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:557, 1927.

9. Claus, H.: Ueber die schwere septische Angina, Verhandl. d. deutsch. Gesellsch. f. inn. Med. **41**:463 (April) 1929.

forms a tonsillectomy after the operation on the neck, if the patient's condition permits. He has obtained from 53 to 70 per cent of recoveries by this method.

Claus [10] called attention to the fact that multiple small infarcts occur in the lungs early in the course of the disease, but are usually not demonstrable by physical signs at this stage. They may extend rapidly within a few days. The tonsils may not show any signs of inflammation, yet on their removal a retrotonsillar abscess may be found. There are a few patients with postanginal pyemia who recover without surgical intervention. In a small group, tonsillectomy alone is sufficient to bring about a cure. Prompt operation is necessary to prevent dissemination of the infection even in comparatively mild cases. Claus' operation consists of exposing the jugular vein from the base of the skull to the clavicle. In addition, the common facial vein and the anterior and posterior facial veins are exposed. The posterior vein is followed up as far as possible to the region of the tonsil. Then, according to the conditions found, either the jugular vein is ligated at the base of the skull and above the clavicle, or only the facial veins (common, anterior and posterior) are ligated. In questionable cases the veins on the opposite side are explored. Where there is only thickening of the fascia or enlargement of the glands, the jugular vein on that side is ligated, while on the apparently normal side, only the facial veins are ligated. The operation on the veins is performed with paravertebral anesthesia and local infiltration; the tonsillectomy, with infiltration of the palatine arch. The tonsils are not infiltrated. General anesthesia is used in children.

Cavernous sinus thrombosis with fatal septic meningitis occurred in two of Claus' cases, owing to retrograde extension of the thrombosis from involvement of the pterygoid plexus and the ophthalmic vein.

In eleven of twelve cases in which autopsies were performed pulmonary infarcts were found in the form of multiple small abscesses, and in several suppurative pleurisy was found. In nine cases there was thrombosis of the jugular vein; in nine cases, thrombosis of the facial vein, and in six cases, thickening of the vascular sheath and pathologic changes in the vascular wall; in four cases there was thickening of the sheath only, without changes in the vascular wall. In all but one case, enlarged glands were found at operation, even though they had not been palpable previously. Bacteriologic examination showed anaerobic hemolytic streptococci most frequently. Staphylococcus aureus and albus were found in one case each. The blood culture was often sterile. In the last six cases that came to autopsy the tonsillar area

10. Claus, H.: Acht und zwanzig Fälle von Pyämie nach Angina, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:114 (May 10) 1929.

was studied with special care, but in only one instance were the small veins in the immediate vicinity of the tonsils found to be thrombosed.

It was found that the course of the disease was mild when the changes in the blood vessels were slight. This was not always so, for in two cases in which there were changes only in the vascular sheath and enlargement of the glands and no changes in the vascular walls, both patients died.

In his last article Claus [11] reported that he had seen ninety-seven cases of pyemia following angina. In his last series of cases (ten) he obtained from 70 to 80 per cent of recoveries.

Bertelsmann [12] reported a case similar to that of Stahl, except that a tender cordlike swelling was present in the left side of the neck. Operation revealed edema of the subcutaneous tissues involving the sheaths of the large blood vessels. The internal jugular vein was freely exposed throughout its length, but there was no evidence of thrombosis in the vein or in any of its tributaries. The wall of the vein appeared somewhat thickened. The cervical glands were somewhat enlarged; all the tissues were edematous, but there was no sign of suppuration. After operation the patient improved rapidly. Culture of the blood before operation was positive for the anaerobic streptococcus. This became sterile after the operation. The jugular vein was not ligated.

Bertelsmann [13] noted that a portion of the internal jugular vein which he obtained from a rapidly fatal case of postanginal sepsis showed no change and no evidence of a thrombus; nor was there microscopic evidence of thrombus formation. The outer coats were hemorrhagic, showing beginning necrosis, and were infiltrated with leukocytes. It is evident that in such cases without thrombus formation changes take place in the walls of the vessels which have not been sufficiently studied.

Drüner,[14] commenting on Bertelsmann's case, stated that the infection usually reached the blood vessels from the lymph channels, and that Bertelsmann had opened up these lymph channels and drained off the infection in his operation to expose the blood vessels.

Waldapfel [15] has reported a large series of these cases. He expressed the belief that venous thromboses arise in a larger number of cases than are discovered at autopsy. These are mostly primary thromboses of

11. Claus, H.: Ueber ein hundert Fälle von Septico-Pyämie nach Angina, Med. Klin. **27**:1269 (Aug. 28) 1931.

12. Bertelsmann, R.: Ueber Postanginöse Sepsis sive Bakterämie, Zentralbl. f. Chir. **54**:642 (March 12) 1927.

13. Bertelsmann, R.: Postanginöse Sepsis und die Lymphbahnen, Zentralbl. f. Chir. **54**:1949 (July 30) 1927.

14. Drüner, L.: Comment on Bertelmann's Case, Zentralbl. f. Chir. **54**:1363 (May 28) 1927.

15. Waldapfel, R.: Post-Tonsillitis Pyemia, Tr. Am. Acad. Ophth. **33**:291, 1928.

the tonsillar vein. He stated that primary thrombosis of the internal jugular vein is also possible. Histologically, abscesses are found in the majority of cases in the immediate neighborhood of the tonsil, while thromboses without abscess formation are almost never seen. The thromboses and bacterial invasion appear to originate from the abscess. Thus, the destructive effect does not come from the tonsil, but from the abscess. The patient's fate does not depend on the condition of the vessels, but on whether or not one is able to overcome the abscess. For this reason, the question of operation on the thrombosed veins is of less importance. Occasionally, there are cases in which no abscess occurs, and in which there is no spreading of the infection along the lymph channels, but in which direct infection occurs by way of the blood stream. In a large majority of the cases of posttonsillar pyemia an abscess occurs in the anterior part of the parapharyngeal space. The abscesses may be multiple and may be connected only by the thrombosed parts of the veins. Another group of cases indicates the possibility of infection of the vein from a suppurating gland. Cases in which direct infection from the blood vessels of the tonsils takes place, without abscess formation, constitute only a small percentage of the examined material.

Waldapfel [16] concluded that recovery ensues without ligation or resection of the vein, when the abscesses can be completely drained. If the abscess cannot be drained completely, the case terminates fatally, even after ligation and resection of the veins. The elimination of the suppurative focus should be the main object of the treatment. All operations on the veins are of secondary importance. He contended that the method of choice consists of drainage of the parapharyngeal space by the external route, after preventive mediastinotomy has been done. If thrombosed veins are recognized, they should be ligated or resected. Operative intervention must come early. This condition rarely occurs in children.

Knick [17] has reported a case in which both the common facial and the posterior facial veins showed thrombophlebitis. The involved veins were ligated and resected. This was followed by an empyema that required bilateral thoracotomy. The patient recovered after a stormy course and a prolonged convalescence. The internal jugular vein was found normal.

Dörfler [18] reported the only case of thrombophlebitis of the internal jugular vein following tonsillitis that he has observed in his forty years

16. Waldapfel, R.: Die postanginöse Pyämie, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:178 (June 10) 1929.

17. Knick: Die operative Heilung der tonsillaren Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:1546, 1927.

18. Dörfler, H.: Beitrag zur Frage der Angina septica, München. med. Wchnschr. **75**:1845 (Oct. 26) 1928.

of practice. Operation was delayed until the development of a pulmonary infarct. The prolonged convalescence might have been prevented had operation been performed earlier.

Burchardt [19] reported a study of numerous sections from the tonsils, tonsillar bed, retrotonsillar tissues, perivascular tissues, lymph glands, tonsillar veins, facial and jugular veins, autopsy material in eleven cases of postanginal sepsis and material obtained at operation in fifteen cases. In all of these cases, perivascular infiltration that could be followed downward to the larger venous trunks was found beginning either in the tonsils or in the tonsillar bed. The lymph glands were always infiltrated and frequently showed evidence of suppuration. In six of the cases there was definite contact infection between the lymph glands and the facial or internal jugular vein, with infective thrombophlebitis of the vein in the immediate region of the point of contact with the lymph glands. The walls of the vessels showed destructive inflammatory changes. In the other five cases definite adhesions between the infiltrated perivascular tissue and the vascular walls were demonstrable macroscopically. In these cases microscopic examination showed destructive inflammatory processes extending from the outer coat of the wall to the media, while the intima was not involved and the lumen was free. In all cases the lymph spaces were filled from the tonsils downward with cells, thrombotic masses and cocci. This supports Uffenorde's theory, because there was no definite evidence of hematogenous dissemination of the infection, macroscopic or microscopic. In no case was there a lesion of the vascular intima only. The important factor in postanginal infection, therefore, appears to be the development of a more or less extensive phlegmonous process that advances by the perivascular route. The lymphatic route also plays a rôle in the extension of the infection. If the perivascular infiltration extends into the vascular walls, it does so either near the primary focus or in the deeper tissues involving the facial or jugular vein. The occurrence of thrombophlebitis is of secondary importance in the production of the angina.

Kissling [20] reported thirty-two cases of postanginal sepsis, and called attention to the fact that sepsis developed in about 2 per cent of all patients with acute tonsillitis who were admitted to his hospital. The blood culture was found positive in twenty-two of thirty cases. An anaerobic streptococcus was found in nine cases. Kissling ligates the internal jugular vein both above and below the point of anastomosis with the facial vein, and slits the latter. A complete tonsillectomy is done after recovery. He has obtained about 50 per cent of recoveries in the patients operated on.

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19. Burchardt, S.: Ein Beitrag zur Pathogenese der postanginösen Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:97 (March 23) 1929.

20. Kissling, K.: Ueber postanginöse Sepsis, München. med. Wchnschr. **76**: 1163 (July 12) 1929.

Joël [21] reported the pathologic findings in cases of postanginal sepsis. No phlebitis was observed in the tonsillar and peritonsillar region, but phlegmonous and suppurative processes were noted in the parapharyngeal tissues. The larger cervical veins were found to have more or less extensive changes in their walls, with adhesive thrombi. The perivascular tissue always showed marked infiltration. In the proximal portion the lumens of the vessels were patent. No changes were found in the veins of the tonsillar bed. This led to the conclusion that the suppurative process spread from the infected tonsils or the peritonsillar tissue through the surrounding loose connective tissue, and then involved the walls of the vessels at some distance from the original process. As a rule, this connective tissue showed edema and a somewhat cloudy fluid, but little frank suppuration. The abscess was not a fulminating process. The danger lay in the involvement of the vascular walls and in the secondary thrombus formation, with resulting sepsis. There were a few cases in which the infecting organisms were present in the small tonsillar veins and from there entered the general circulation, but these cases were not frequently found. This author concluded that the blood vessels are secondarily involved in a majority of cases. He found no evidence that the infecting organisms enter the circulation by passing through the lymph channels and that the lymphatic involvement is secondary. His view corresponds in general with that of Uffenorde, except that he considers the lymphatic route of less importance than does Uffenorde. His findings do not support the theory of Fränkel, that the thrombophlebitis in these cases is primary.

Dixon and Helwig [22] reported six cases of thrombophlebitis of the jugular vein after acute tonsillitis, with recovery in two cases. All occurred in female patients. Autopsy showed multiple abscesses of the lungs in three; thromboses were found as follows: cavernous sinus, one; jugular bulb, two; lateral sinus, one. They mentioned that Goodman, in 1917, resected the jugular vein for thrombophlebitis following tonsillitis, with recovery.

Reye [23] reported thirty similar cases, with fourteen recoveries. He spoke of two types of systemic infection following angina: In the acute form, the symptoms of general infection develop while the tonsillar abscess persists without the temperature returning to normal. There are chills and an increase in fever, with signs of jugular thrombosis and metastases. The second is a more chronic form, in which the

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21. Joël, W.: Ueber die postanginöse Pyämie und ihre Verbreitungswege, Deutsche med. Wchnschr. **55**:2133 (Dec. 20) 1929.

22. Dixon, O. J., and Helwig, F. C.: Thrombophlebitis of the Internal Jugular Vein as a Complication of Tonsillitis, Ann. Otol., Rhin. & Laryng. **39**:1137 (Dec.) 1930.

23. Reye, E.: Zur Pyämie nach Angina, Med. Welt **4**:1791, 1931.

tonsillar process entirely subsides and the tonsils appear normal, or only slightly enlarged. The temperature may become normal, or there may be some fever from time to time and slight pain at the angle of the jaw. Chills and a sudden rise in temperature develop, and various metastatic lesions occur. Bacteriologic examination showed anaerobic streptococci in fifteen cases, anaerobic bacilli in one case, pneumococci in one case and hemolytic streptococci in twelve cases. Twelve of the sixteen patients with anaerobic streptococci and the one patient with anaerobic bacilli died. Ten of the twelve patients with hemolytic streptococci recovered. The anaerobic organisms were therefore the most dangerous and were associated chiefly with chronic abscesses.

Myerson [24] reported a case of septicemia and death following streptococcic tonsillitis in 1921. He stated that such cases probably occur more frequently than is generally supposed, and he called attention to the necessity of knowing the offending organism in cases of acute tonsillitis. He stressed the fact that complications more frequently follow a streptococcic infection than other types. He did not speak of anaerobic organisms, however.

Eagleton [25] has had four cases of the type being discussed. Two of the patients recovered. He expressed the belief that the infection travels from the tonsillar veins into the jugular veins. He exposes the tonsillar veins, removes the infection in the pterygomaxillary fossa and ligates the jugular vein.

Mosher,[26] in a recent article, stated, "Thrombosis of the internal jugular vein still takes its weekly toll under the guise of septicemia of unknown origin."

SUMMARY AND CONCLUSIONS

The recent literature on the subject of pyemia following acute tonsillitis has been reviewed.

1. Pyemia following acute tonsillar infections is not a rare condition and probably occurs in from 1 to 2 per cent of such cases. A chill or series of chills, occurring from two to three days after the beginning of a sore throat, or one occurring a week or two after an angina that has subsided, with a swelling along the anterior part of the sternocleidomastoid muscle or over the parotid region, may be the beginning of pyemia. One must exclude pneumonia, pyelitis or one of the blood dyscrasias, such as agranulocytosis or leukemia. Once the diagnosis is established, immediate exploration of the neck is advisable.

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24. Myerson, M. C.: Septicemia and Death Following Streptococcic Tonsillitis, Laryngoscope **31**:847, 1921.

25. Eagleton, W. P.: Personal communication to the author.

26. Mosher, H. P.: Submaxillary Fossa Approach to Deep Pus in the Neck, Tr. Am. Acad. Ophth., 1929, p. 19.

2. The phlebitic process may extend downward from the tonsillar veins and involve the internal jugular and facial veins or may extend in the opposite direction to reach the cavernous or lateral sinus. When the cavernous sinus is involved, the infection spreads by way of the pterygoid plexus or the petrosal sinuses. Nearly all of the autopsies have shown the presence of pulmonary infarcts or abscesses. Pleurisy, pneumonia or empyema following acute tonsillitis with a history of chills may be a manifestation of a pyemic process. It has been demonstrated that thrombi are present in the tonsillar veins in nearly all cases of acute tonsillar or peritonsillar inflammation. It is possible that in some cases abscesses of the lungs occurring after tonsillectomy may be explained on the basis that a tonsillectomy is done before the inflammation of the tonsil has subsided. The trauma resulting from the operation causes the phlebitic process to spread. In many cases the inflammation in the tonsil may not be demonstrable.

3. There is a difference of opinion as to the manner in which the infection spreads from the tonsillar area. Fränkel thinks that it reaches the jugular vein by way of a thrombophlebitis of the tonsillar veins, while Uffenorde contends that the infection spreads along the lymph channels to the internal jugular vein and other veins outside of the immediate region of the tonsillar focus. Burchardt and Joël believe that it travels by way of a perivascular process in which the blood vessels are secondarily involved. Waldapfel's impression is that the abscess in the tonsillar region is the principal focus and that infection spreading through lymph channels or blood vessels is of minor importance; he believes that the destructive effect does not come from the tonsil, but from the abscess.

4. The anaerobic streptococcus is the organism most frequently found. Blood cultures are positive in about 50 per cent of the cases. The presence of anaerobic streptococci or bacilli in the blood stream should make one consider the possibility of a preexisting tonsillar infection as the cause. In two of our cases the blood culture was reported positive. The prognosis appears to be more grave when such organisms are present.

5. I suggest that the Tobey-Ayer test should be used to determine on which side the jugular thrombosis is present. In one of my cases (case 3) with an otitis on the opposite side, it made the diagnosis positive.

6. There is no agreement as to the management of these cases. Claus suggests an exposure of the structures of the neck from the mastoid process down to the clavicle. The parapharyngeal space should be exposed, and any infection found should be dealt with. Ligation or resection of the jugular and facial veins is also carried out. If it is

impossible to determine on which side the infection is taking place, he suggests exposing the neck on both sides, and if the jugular vein is involved on one side, he suggests ligating or resecting it and ligating and resecting the facial vein on the opposite side. Waldapfel opens the parapharyngeal space and evacuates the abscess, at the same time protecting the mediastinum. Kissling obtains good results by simply opening the parapharyngeal space and draining the abscess. He ligates the jugular vein above and below the facial vein and opens the latter. In some cases the thrombus may extend to the jugular bulb or even upward so that it may become necessary to open the mastoid process to remove the sinus plate and expose the lateral sinus. Meningitis may be caused by involvement of the jugular bulb and the lateral sinus in this condition.

7. In every case of sepsis following angina, the parapharyngeal space should be explored. There is a difference of opinion as to whether the tonsils should be removed at the time of operation. Some men advocate their removal if the condition of the patient permits; others wait until a favorable time after recovery.

Four cases of pyemia following acute tonsillitis are recorded, with recovery in one.

The patients in cases 1 and 2 had a swelling over the parotid region.

The patient in case 3 had a complicating chronic otitis media on the right side and an extensive jugular thrombosis on the left.

Autopsies were performed in cases 1 and 2, and in both multiple abscesses of the lungs were found.

Case 2 showed a thrombus in the lateral sinus.

In case 4, in which the patient recovered, the internal jugular vein was not ligated.

201 Eastern Parkway.

CHANGES IN THE NASAL ACCESSORY SINUSES AFTER BIRTH

W. WALTER WASSON, M.D.
DENVER

The antrums and ethnoids are the most fully developed of the nasal accessory sinuses of the full term fetus. At the time of birth, these sinuses are of appreciable size and fairly well surrounded by bony or cartilaginous structures. Later they contain air, and one usually thinks of them as cavities filled with air. However, in the unruptured amniotic sac these sinuses cannot contain air, and they only become aerated some time after birth. What then are the contents of the sinuses while in the unruptured amniotic sac, or during the period of birth before aeration has taken place? The time necessary for this clearing process to take place together with the physiologic phenomena involved constitutes a problem of rather unusual interest. The influence which variations of the physiologic phenomena may have on the subsequent health of the infant makes this study of considerable importance. The same problems apply to the middle ear and the mastoid, but at this time, I wish to consider only the ethmoids and the antrums.

A rather careful survey of the literature reveals no information on such a study of the sinuses. Griffith and Mitchell [1] stated that the middle ear contains no air at birth but only swollen mucous membrane and mucus, and von Reuss [2] said that at birth the tympanic cavity contains only a yellow fluid and gelatinous tissue. But no reference could be found which dealt with these problems as they relate to the nasal accessory sinuses. There are several textbooks which give detailed descriptions of the anatomy of the sinuses. Schaeffer [3] stated that "Even though the maxillary sinus is a cavity of considerable size in the newborn, often measuring 8 by 4 by 6 mm., it is not always demonstrable in skiagrams. This is probably due to the slit-like shape of the sinus,

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Read before the Section on Radiology at the Eighty-Third Annual Session of the American Medical Association, May 13, 1932.

From the Child Research Council and the University of Colorado School of Medicine.

1. Griffith, J. P. C., and Mitchell, A. G.: The Diseases of Infants and Children, ed. 2, Philadelphia, W. B. Saunders Company, 1927, vol. 1.

2. von Reuss, A. R.: The Diseases of the Newborn, New York, William Wood & Company, 1922.

3. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

and the fact that it hugs the lateral wall of the nasal fossa closely." Funkhouser and McAliley [4] said, "It would seem that a cavity 8 by 4 by 6 millimeters should be demonstrable on an x-ray photographic plate, but frequently it is impossible." This is the consensus of all the writers whose books or articles were reviewed, and as far as the discussion is carried.

The anatomic descriptions in such texts as Hajek [5] are most excellent. They deal with the embryologic formation and the morphology of the sinuses and their sizes at various prenatal and postnatal periods. For further study, I refer the reader to these texts. I wish to emphasize the fact that the maxillary antrums are formed by a pouch growing into the maxilla from the middle meatus, and in a full term fetus this

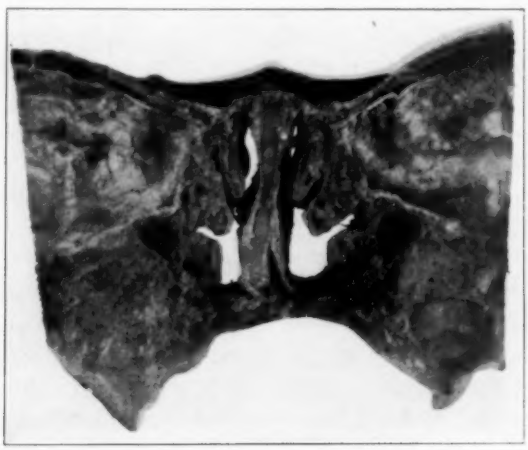

Fig. 1.—Sagittal section through the head of a full term fetus, showing the maxillary antrums, turbinates and nasal fossas. Note the position of the antrum and its relation to the floor of the nose, the inferior turbinate and the orbit of the eye.

sinus lies directly lateral to the inferior turbinate. Therefore it is, in a general way, surrounded by the maxillary bone on the lateral side and the bony and cartilaginous structures of the inferior turbinate on the medial side (fig. 1). In the dried and disarticulated skull it appears as a small depression in the maxilla (fig. 2). Its median diameter is 14 mm. dorsal to the anterior nasal spine of the maxillary bone. The average diameters of the maxillary sinus of a full term fetus are given

4. Funkhouser, W. L., and McAliley, R. G.: Diseases of the Respiratory System in Infants and Children, in Haynes, R. S.: Clinical Pediatrics, New York, D. Appleton and Company, 1927, vol. 9.

5. Hajek, M.: Pathology and Treatment of the Inflammatory Diseases of the Nasal Accessory Sinuses, St. Louis, C. V. Mosby Company, 1926.

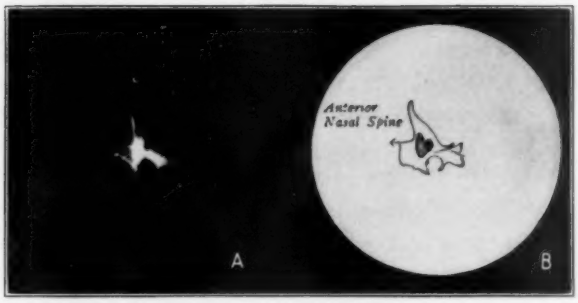

Fig. 2.—*A*, postero-anterior roentgenogram of the dried maxilla of a full term fetus, showing the antral depression which has been filled with barium. *B*, sketch of the same. The shaded area denotes the antral depression.

Fig. 3.—*A*, roentgenogram of the dried facial bones of a full term fetus, showing the relation and appearance of maxilla and turbinates. *B*, sketch of the same. Note the position and size of the infra-orbital canal which is sometimes mistaken for the antrum.

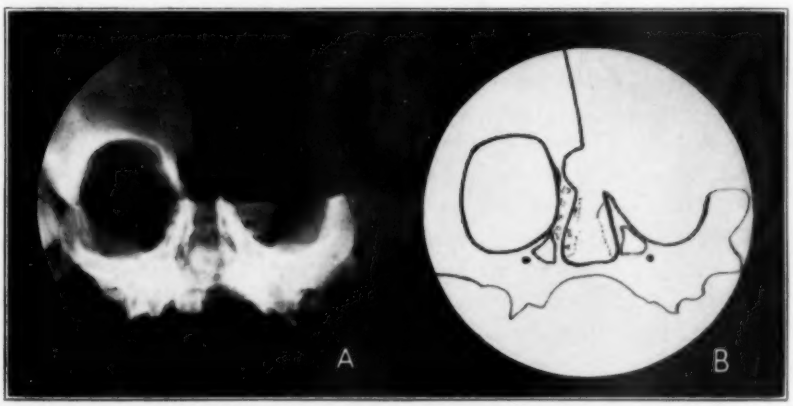

Fig. 4.—*A*, roentgenogram of the dried, partially reassembled skull of a full term fetus, showing the relation of turbinates, antrums, ethmoids and infra-orbital canals, in the postero-anterior position. *B*, sketch of the same.

by Schaeffer [3] as: ventrodorsal, from 7 to 8 mm.; mediolateral, from 3 to 4 mm., and cephalocaudal, from 4 to 6 mm.

The ethmoidal sinuses in the dried skull are found to lie in the ethmoid bone and each one is surrounded by a delicate osseous structure. Their sizes in the full term fetus are given by Schaeffer [3] as: anterior group, 2 by 2 by 5 mm.; posterior group, 2 by 4 by 5 mm. Both the ethmoids and the antrums are lined with mucous membrane and both have openings into the infundibulum ethmoidalis. It is thought that the ethmoidal cells also have separate ostia. The ethmoidal cells lie directly cephalad and medial to the antrum (fig. 3).

As just pointed out, a survey of the literature reveals nothing concerning the physiology of the antrums and ethmoids during the period of birth. Likewise, there is no available information as to the influence

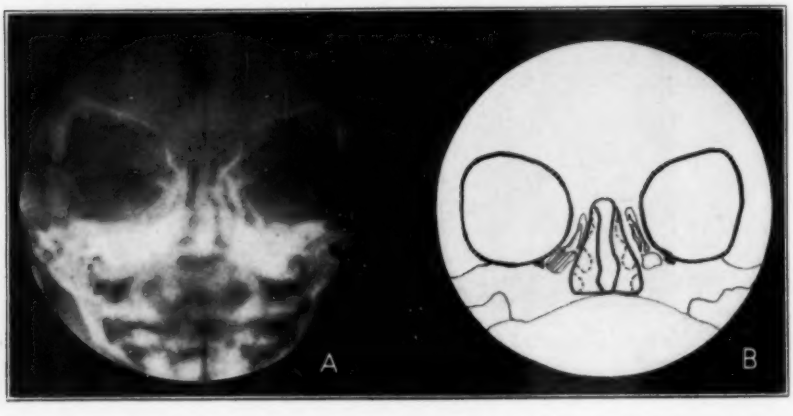

Fig. 5.—*A*, postero-anterior roentgenogram of head of boy, aged 4 days, showing aeration of the left antrum, but not of the right. *B*, sketch of the same. The shading indicates increased density of the right antrum.

which a deranged physiology of these sinuses would have on the subsequent events of the life of the infant. It is noteworthy that Schaeffer [3] stated, "in some cases, the cleft of communication between the maxillary ostium and the meatus nasi medius is practically shut off . . ." As regards infection, the respiratory tract, that is, the lungs, throat, sinuses, ears and mastoids, must be considered as essentially one cavity.[6] There are many reported cases of infection of the sinuses of the infant even in the first few weeks of life and many cases of abnormalities, but there is nothing in the literature on the beginnings of disease in these structures. Formerly, it was taught that there was no infection of the sinuses in the first few months of postnatal life, and even today the importance

6. Wasson, W. W.: Bronchosinusitis Disease, J. A. M. A. **93**:2018 (Dec. 28) 1929.

of infection during this period is underestimated. Respiratory infections are the most serious of all diseases during the first decade of life, and a disordered physiology of any one part of the respiratory system can well play an important part in the origin of such respiratory diseases. I wish to point out that only by longitudinal studies can one have a visual conception of the real situation, as the infant grows and develops from month to month. In such longi-

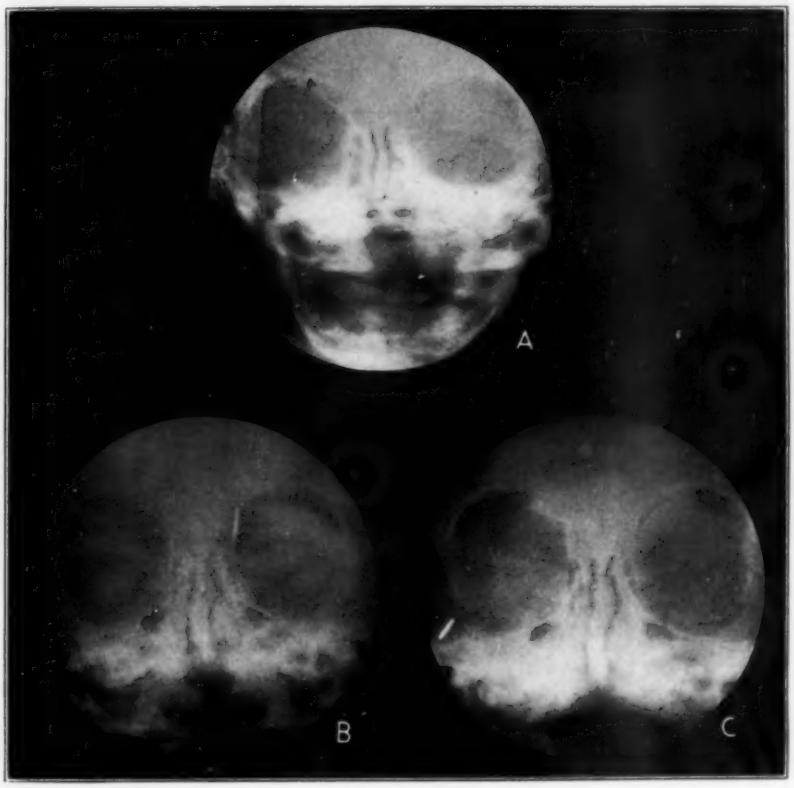

Fig. 6.—Postero-anterior roentgenograms of the head of a girl: *A*, taken at 3½ weeks of age, sinuses all dense and showing no aeration; *B*, at 8 weeks of age, showing bilateral clearing; *C*, at 6 months of age, showing growth and bilaterally clear sinuses.

tudinal studies, roentgen examination reveals in the living infant the anatomic growth and some of the physiologic and pathologic processes. The statement made so often in textbooks, that roentgenograms of the sinuses are unsatisfactory in early childhood, should be discontinued.

My interest in the nasal accessory sinuses during the period of birth was occasioned by longitudinal studies of a series of infants from birth to 10 years of age. In reviewing this series, I observed that the sinuses

of infants less than 2 years of age frequently showed a density of one or more of the antrums or ethmoids. It was noted further that at 1 month of age there was no aeration of the sinuses. This was rather an unexpected observation, and it necessitated a more thorough investigation of this period of life. The aeration of the sinuses seemed to require longer than I had expected, and likewise seemed to be associated with or have some influence on subsequent events. It was decided to

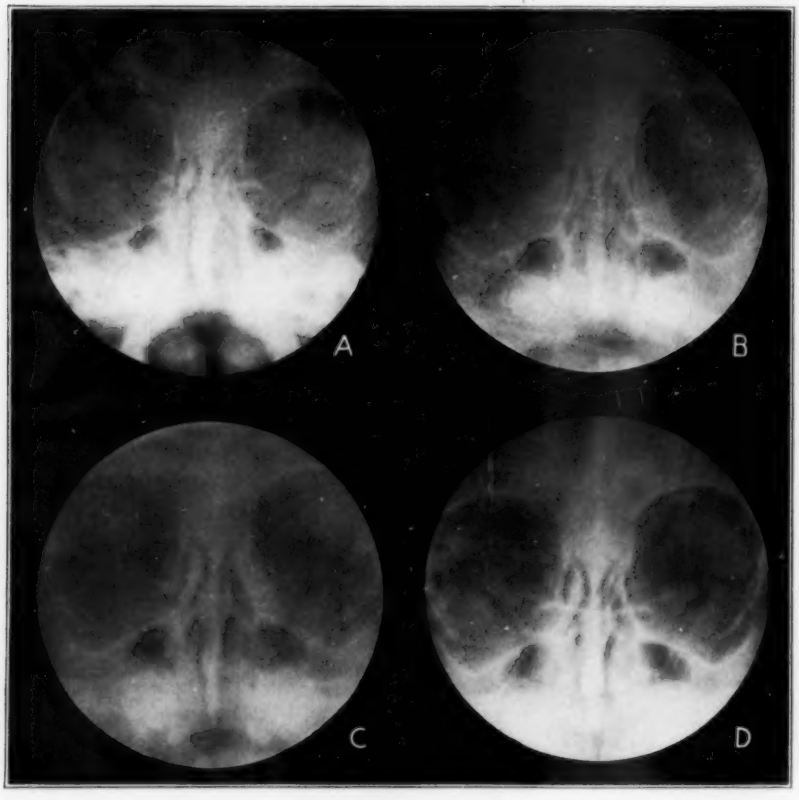

Fig. 7.—Postero-anterior roentgenograms of the same patient as shown in figure 6, showing continued bilateral clearness of the sinuses: *A*, taken at 9 months of age; *B*, at 12 months; *C*, at 16 months, and *D*, at 18 months.

approach the problem by endeavoring to obtain information on the following points: 1. When do the sinuses become fully aerated? 2. What are the contents of the sinuses before and immediately after rupture of the amniotic sac? 3. What factors influence the aeration of the sinuses? 4. What influence does the complete or incomplete aeration have on subsequent events in reference to the respiratory tract?

In this paper, I wish to attempt a discussion of the first and fourth points just outlined. Investigation of the second and third points has

been assigned particularly to the department of pathology under the supervision of Dr. William C. Johnson. It is evident that the roentgenogram would be of greatest value in the approach to the first and fourth parts of the study and a knowledge of the anatomy a first requisite.

A partial review of the anatomy of the ethmoids and antrums has been given. In the examination of the dried bone by means of roentgenograms, no definite antrum could be shown. A few spaces were

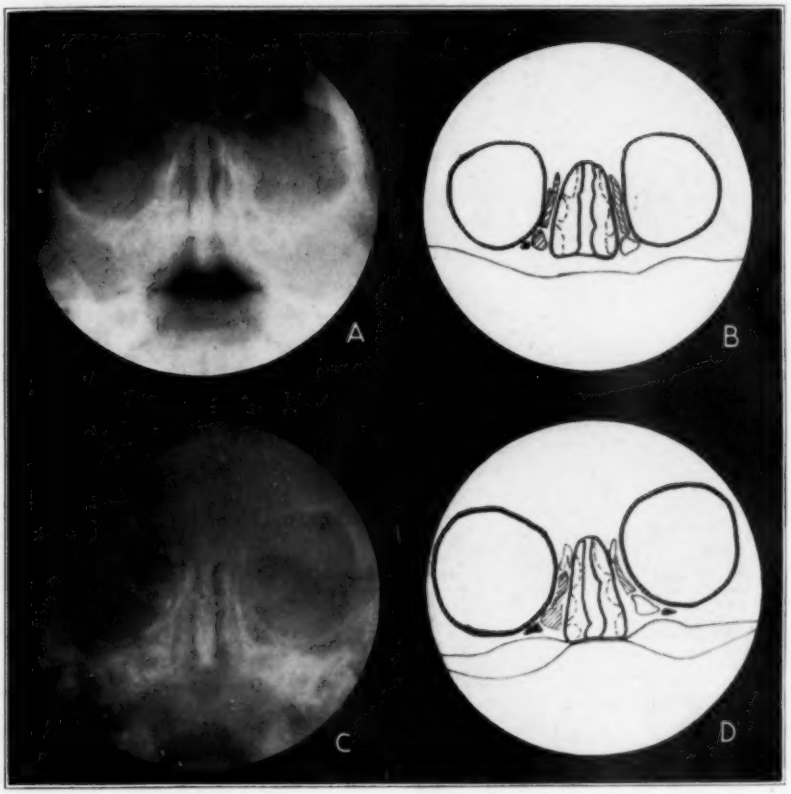

Fig. 8.—Postero-anterior roentgenograms of the head of a girl, showing the left antrum partially aerated. *A*, taken at 3 weeks of age; *B*, sketch of the same, the shading indicating density of the sinuses; *C*, taken at 7 weeks of age; *D*, sketch of the same.

observed in the region of the ethmoids, but again these were rather indefinite. It is to be noted that in the dried bony specimen the antrum is merely a depression of the bone and not a true cavity as one would expect in the living in whom the antrum is completely surrounded by tissue. This part of the study, therefore, chiefly concerned itself with the location of the antrum and the ethmoids and their appearances at this age (fig. 4).

Roentgenograms were then made of a series of infants from the department of obstetrics at 4 and 9 days of age. (These babies leave the hospital on the tenth day.) Of forty-seven babies there was evidence of aeration of the sinuses in only eight. Of this number, the left antrum was aerated in five and the right antrum in three. In no case was there complete bilateral aeration. From this study one may conclude that there is only incomplete aeration of the sinuses during the first ten days

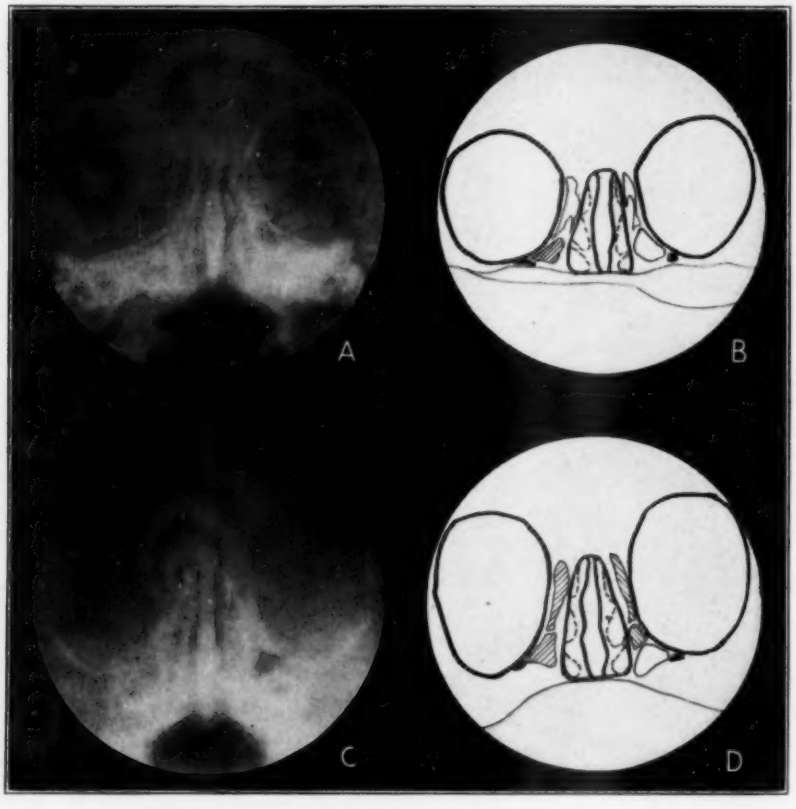

Fig. 9.—Postero-anterior roentgenograms of the same patient as shown in figure 8, showing aeration of the left antrum. *A*, taken at 3½ months of age; *B*, sketch of the same; *C*, taken at 5 months; *D*, sketch of the same. Note that the right antrum remains dense.

of life, and this occurred in only 17 per cent of the cases. The conclusion assumes that air within the sinuses can be portrayed as was seen in some of the cases (fig. 5).

As stated before, there was a longitudinal series available in which roentgenograms had been made from as early as the thirteenth day and at intervals of from one to three months on the same child throughout the first decade. This series in which roentgenograms of the sinuses

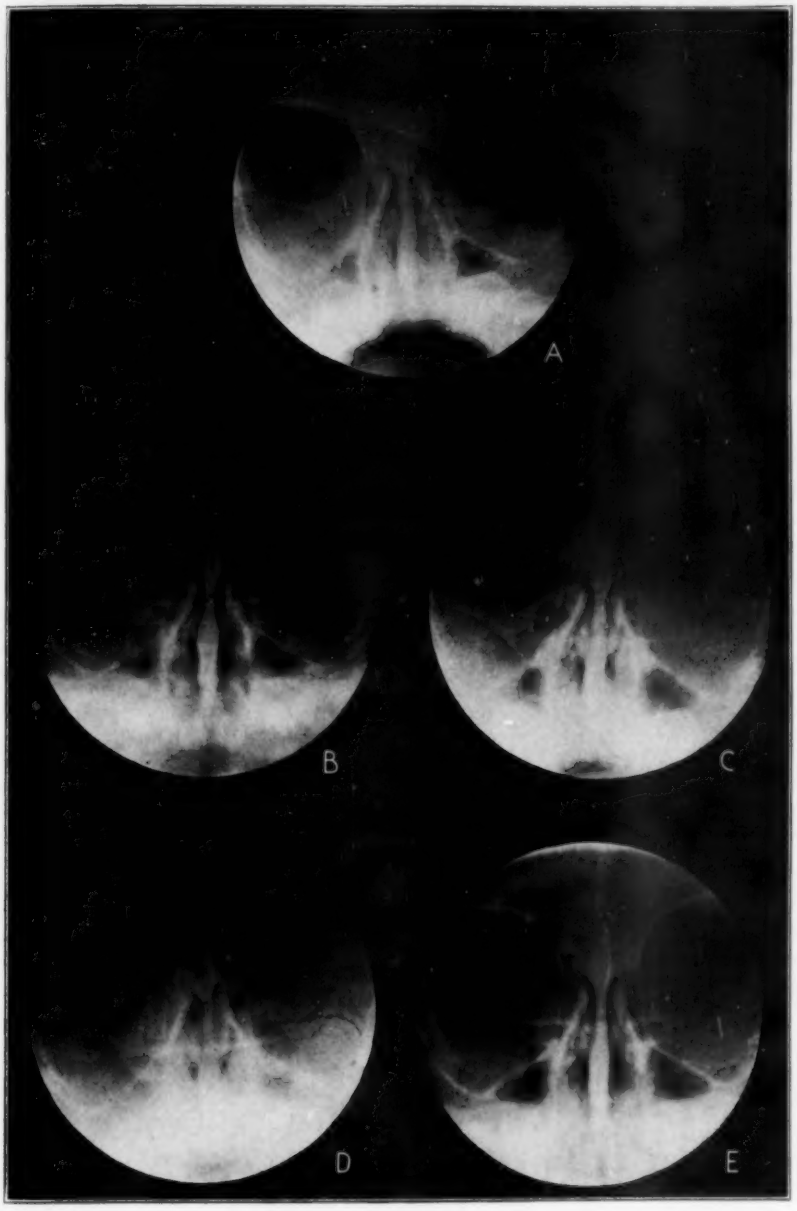

Fig. 10.—Postero-anterior roentgenograms of the same patient as shown in figures 8 and 9, showing continued unilateral (left) antral clearness and partial density of the right antrum: *A*, taken at 8 months of age; *B*, at 10½ months; *C*, at 12 months; *D*, at 14½ months, and *E*, at 18 months.

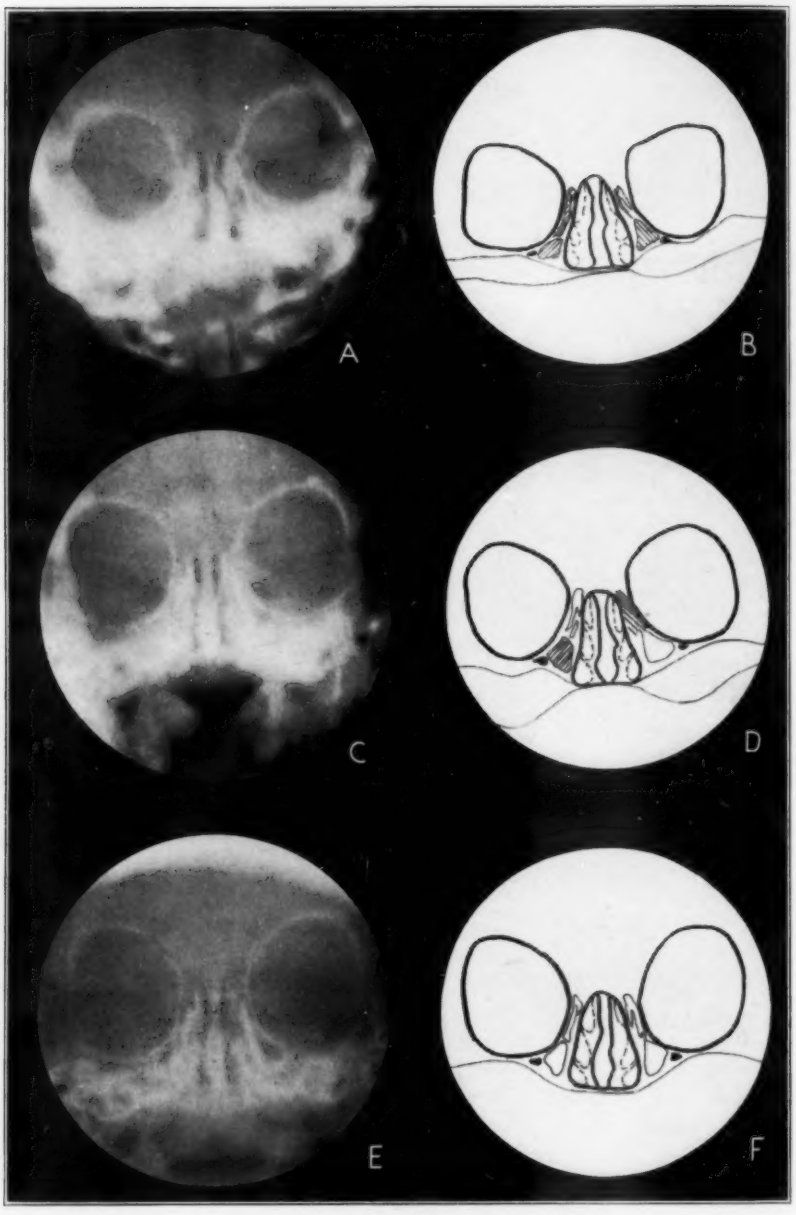

Fig. 11.—Postero-anterior roentgenograms of the head of a girl: *A*, taken at 13 days of age, showing no aeration of any of the sinuses; *B*, sketch of the same, the shading indicating density of the sinuses; *C*, taken at 6 weeks of age, showing partial aeration of the left antrum; *D*, sketch of the same; *E*, taken at 2½ months of age, showing bilateral, fairly complete aeration of ethmoids and antrums; *F*, sketch of the same.

were made during the first two months totaled thirty-eight cases. In reviewing this series, it was observed that they grouped themselves into three classes: (1) those infants in whom there was complete bilateral aeration of the antrums and ethmoids; (2) those in whom there was unilateral aeration, and (3) those in whom there was no aeration of either side as evidenced by bilateral persistent density of the sinuses.

Since roentgenograms were made of many of these infants under 1 month of age, it was possible to gather some conception as to the period

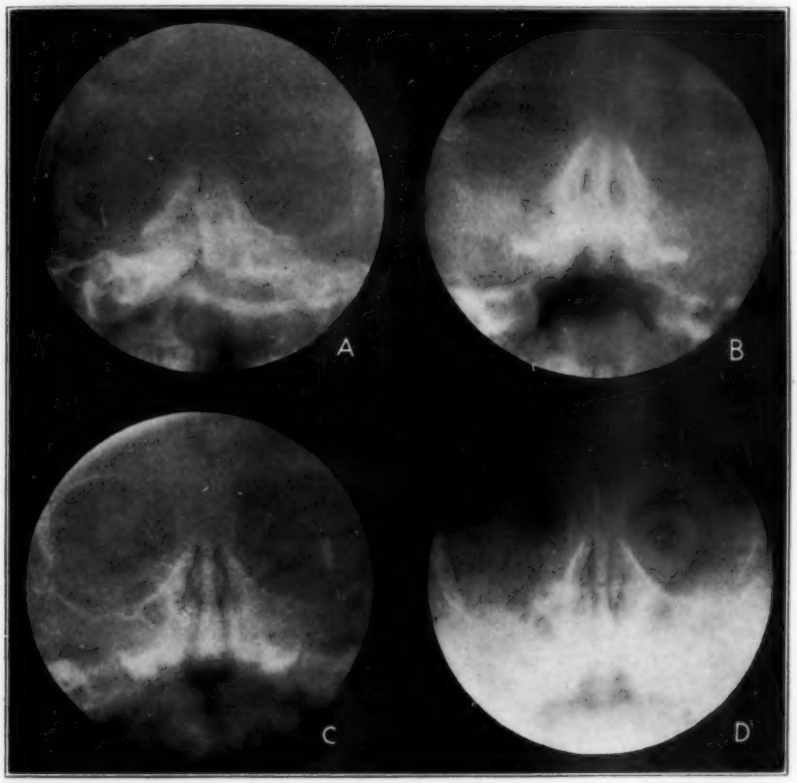

Fig. 12.—Postero-anterior roentgenograms of the head of a girl, showing bilateral density of the maxillary antrums: *A*, taken at 2 months of age; *B*, at 6 months; *C*, at 9 months, showing partial clearing of the right antrum, and *D*, at 12 months, showing partial clearing of both antrums.

of time necessary for the aeration of the sinuses. At 1 month of age, there were two infants who showed bilateral aeration, five who showed unilateral aeration and thirty-one who showed no aeration. At 2 months of age, there were one infant who showed bilateral clearing, eight who showed unilateral clearing and thirteen who showed no aeration. It was definitely noted that if the sinuses cleared bilaterally, they remained clear

throughout the first two years of life (figs. 6 and 7). On the other hand, if there was unilateral clearing, then that condition persisted consistently during the first two years (figs. 8, 9 and 10). If bilateral clearing took place following aeration of one side only, and later an increased density became apparent, the side which had been slow to become aerated showed the greater increased density (fig. 11). It is apparent that the density of the sinuses in question, in the early weeks of life, could not be said to result from infection, and

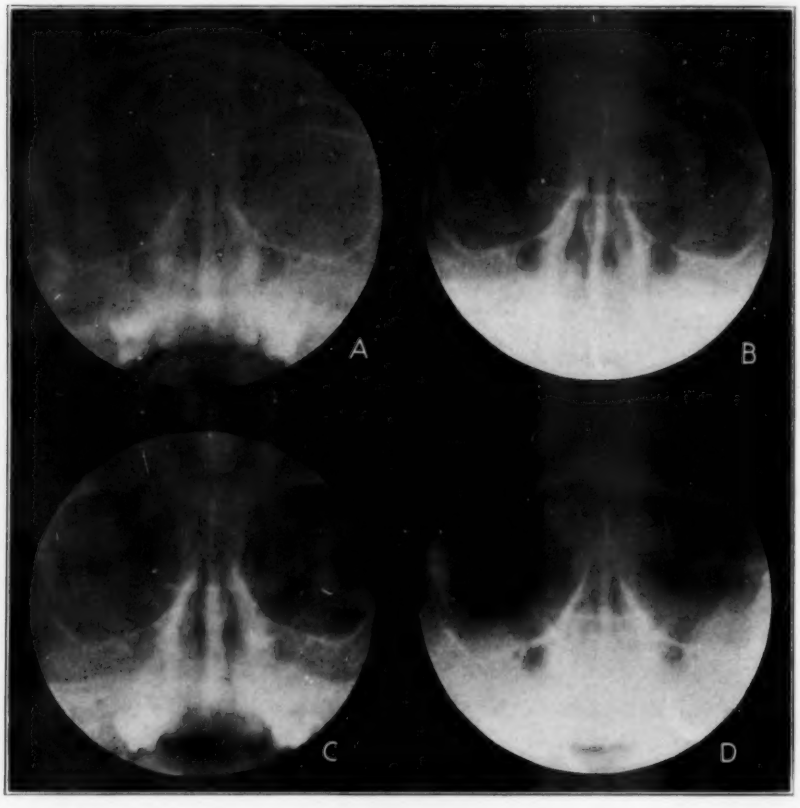

Fig. 13.—Postero-anterior roentgenograms of the same patient as shown in figure 12, showing continued partial clearing of the maxillary antrums. Note that when there is an increased density of the sinuses both antrums show this increased density as in *C*. *A* was taken at 15 months of age; *B*, at 18½ months; *C*, at 22 months, and *D*, at 24 months.

probably not from allergic responses. In the later part of the two year period under discussion, some of these densities were no doubt due to infection, and it was observed that the sinus slow in becoming aerated showed the greater involvement. In some infants in whom there was no aeration of either side this condition persisted with certain variations

throughout the first two years and developed into frank infection of sinuses (figs. 12 and 13). One would conclude from this series that in general the antrums and the ethmoids of an infant do not become fully aerated until from four to eight weeks after birth, and that the state of aeration is an important influence on the condition of these sinuses during the first two years of life.

SUMMARY

Although several years have been given to the collection and study of the material just presented, this paper must be considered as a preliminary report. No definite conclusions are advisable at this time, but certain comments are offered.

1. In spite of the appreciable size of the antrums and ethmoids of the new-born infant, aeration of these sinuses takes place slowly.

2. The first two months of life constitute an important period, and the physiology of the sinuses during this period is not understood.

3. When observed by longitudinal studies, the deranged physiology of the sinuses during the first few weeks of the new-born would seem to have important influences on subsequent conditions of the sinuses.

4. The greatest care must be exercised in making a diagnosis from roentgenograms of the nasal accessory sinuses, during the early weeks of life.

ABSTRACT OF DISCUSSION

DR. AMÉDÉE GRANGER, New Orleans: This splendid presentation brings up some interesting points indeed and reminds one of the work done jointly by the otolaryngologist and the pathologist, which seems to prove, at least to my mind, that an early infection of the sinuses will prevent later pneumatization and development. It is not only possible but highly probable that infection of the sinuses may exist very early in life, and, as so well brought out by Dr. Wasson, this will explain the lack of development or the lack of pneumatization of the sinuses in later life.

The impression several years ago was that the sphenoid sinus did not develop until about the twelfth or thirteenth year. I am quite sure that Dr. Wasson will bear me out when I make the statement that as a matter of fact the sphenoid develops very much earlier than that. Pneumatization has been seen to start as early as the second year. It is interesting to note, however, that even when the pneumatization is not visible on the lateral roentgenogram of the head the sphenoid line (Granger line) is present; it has been possible to diagnose infections of these infantile sphenoids by the absence of that line and a definite increase in the opacity of the sphenoid region on the affected side.

DR. EDWIN C. ERNST, St. Louis: Dr. Wasson's interesting work and his methods of investigation are indeed most commendable. Greater effort should be expended in the direction of more intensive and detailed systematic study of the developmental phases of our problems. Therefore, this type of research should prove to be of inestimable value in the future interpretation of the adult sinus diseases on the basis of early sinus development or possible infections.

The one point I should like to emphasize is that Dr. Wasson's work is a stepping stone to better roentgenologic diagnosis of sinus disease. The differential diagnoses of undeveloped or infected sinuses in children is frequently most important and difficult even though the age is given due consideration. I should like to ask if there are any differential diagnostic points of value other than the age factor?

As to the use of radiopaque oils in children, I certainly believe that Dr. Wasson is in a good position to take up this work. It might prove of inestimable value. Occasionally, the insufflation of sinuses, as advocated by Dr. Proetz, by means of radiopaque oils is of value therapeutically.

Of course, the difficulties in developing such a technic for use in children may be insurmountable.

At any rate, we must not allow ourselves to be placed in an unfavorable position as consultants but must insist on taking advantage of the full value of all that roentgenology has to offer and thus help our colleagues solve their sinus problems.

Dr. Eugene P. Pendergrass, Philadelphia: I was much interested in Dr. Wasson's work on the infant's maxillary sinus. Three or four years ago my associates and I studied a series of twenty-five infants, taking roentgenograms at 2, 7 and 12 days of age. We found absence of pneumatization in some of these cases, but our percentage of pneumatization even bilaterally was much higher than in the group presented this morning. In the interpretation of disease, we used the appearance of a nasal cavity to help us. Encroachment on the nasal cavity often occurs in disease of the maxillary sinus in infants. It helped us in the interpretation of one case in which there was osteomyelitis of the maxilla in a child 18 days of age. I am afraid that we would not have called attention to this, other than just a casual remark, if I had known about Dr. Wasson's work. We have seen nonopaque foreign bodies in children that show occluding of the maxillary sinus and an encroachment on the nasal cavity as early as 4 months of age. One should keep in mind, too, that congenital conditions such as stenosis of the posterior areas do occur in children not infrequently.

Dr. Bundy Allen, Tampa, Fla.: Dr. Wasson's paper is most interesting and, no doubt, will prove to be very instructive. From the observation of his slides, I feel that some of these sinuses are infected and not blurred as a result of the lack of pneumatization. One may find on further investigation that many of these sinuses, as shown in children, are infected. Dr. Wasson said that he would not make a definite statement, which leaves the matter open for further study. Therefore, I feel that we should all continue the study of these sinuses in some such manner as has just been outlined by Dr. Wasson.

Dr. W. Walter Wasson, Denver: In spite of the fact that many years have been spent in collecting the material and studying the longitudinal series on which this paper is based, I presented it as a preliminary report. The longitudinal series is made up of children from all social stratas. The records date from the birth of each child and are carried throughout his life. Each child is examined physically, roentgenographically, psychologically and clinically at regular intervals. The study of a group of cases at any one "age period" in an endeavor to construct the rest of the child's life history from that isolated material is different from building a conception of the development of the child from the time of birth by the longitudinal method of study. Ideas and conceptions formulated from the study of a single age group often prove inadequate and erroneous in comparison with information gained from the progressive study of a constant group of children growing under the observers' eyes. Perhaps, in this paper, I have given the impression of being ultra-conservative. If you had pondered over this problem as much as I, and had

watched the evolution of various ideas, and had listened to the conjectures as to what it might all mean, you, too, would be conservative. Dr. Eugene Pendergrass and Dr. Bundy Allen have spoken of the possibility of these sinuses being infected. That has been considered. But it is certain that the hundred babies reported here were all average, normal babies. They had no clinical evidence of infection.

There is a definite problem of which I wish to speak. That is, there is a clearing process of the sinuses which takes place after the delivery of the baby. The question then is: How long does that clearing process require? Dr. Pendergrass suggested the possibility that working at a lower altitude, he might find a different percentage from mine which was made at an altitude of one mile. True, atmospheric pressure may play a part; seasonal variations may play a part, and likewise conditions in the nursery and anomalies must be considered. Schaeffer suggests that the ostium of the antrum may be practically occluded in many cases. Each of the foregoing factors may play a part in this problem, and the consideration of their potential influences presents many reasons for being conservative. Therefore, it would be premature for me to make a definite statement at this time.

That there is a close relation between the clearing process and the conditions, pathologic or otherwise, that take place afterward, is well shown by my series of cases. It is known that the clearing process has an influence. What this influence is and how much it means are the questions which I hope can be worked out. Not just one longitudinal series must be studied, but many. There must be similar observations by different investigators working all over the world, at different altitudes, in the various seasons, with children from all social stratas, and with normal as well as pathologic conditions. At the present time in Denver, my associates and I are studying a series of stillborn infants and a series of new-born infants that die by sectioning the sinuses and taking roentgenograms of them. This is just another phase of the problem, and another possible approach to its solution. There is no doubt in my mind that the whole problem will require considerable further study from many different angles.

VIDIAN NEURALGIA

WITH SPECIAL REFERENCE TO THE EYE AND ORBITAL PAIN IN SUPPURATION OF THE PETROUS APEX

HARRIS H. VAIL, M.D.
CINCINNATI

The real title of my paper should be this question, "Is the ocular pain typical of cases of suppuration of the apex of the petrous bone due to an irritation of the ophthalmic division of the fifth nerve?" This question may seem to be one raised through extreme ignorance. One has only to read the recent articles of Eagleton, Kopetzky and Almour, Profant, Friesner, Druss and Ramadier to conclude that the opinion is held by most authors that the ocular and orbital pains so typical of suppuration of the petrous apex are due to an irritation of the ophthalmic division of the gasserian ganglion.

Ramadier,[1] who noted, as have others, the typical location of the ocular pain, asked why only the ophthalmic division should be affected when the focus of osteitis was in relation to the other parts of the trigeminal tract. He felt that the question up to now had not been answered satisfactorily.

If one does a little serious thinking, one may wonder why there should be a selective distribution of pain when presumably the entire nerve trunk has been irritated. If the pain in the back of the eye is due to an irritation of the ophthalmic nerve by stretching or being held down by adhesions to the dura, as Eagleton [2] feels, why should the pain remain confined to a relatively small portion of the area supplied by the nerve? It would seem logical to believe that if a nerve is irritated, the stimulus should pass over its entire distribution. Thus far my argument is purely hypothetic, and before going further into it I should like to answer the question raised at the beginning of my article as follows: "The ocular and orbital pain in cases of suppuration of the apex of the petrous bone is not due to irritation of the ophthalmic division of the fifth nerve."

Read at the Sixty-Fifth Annual Meeting of the American Otological Society, Atlantic City, N. J., May 17, 1932.

1. Ramadier, J.: L'ostéite profunde du Rocher, Ann. d'oto-laryng. **50**:1300 (Dec.) 1931.

2. Eagleton, W. P.: Sixth Nerve Involvement in the Diagnosis and Surgical Treatment of Meningitis of Otitic and Nasal Origin, Congr. internat. d'oto-rhino-laryng., Copenhagen, 1928, p. 824.

Kopetzky and Almour [3] merely mentioned the great superficial petrosal nerve.

Eagleton [4] stated that irritation of the superficial petrosal of the facial nerve may cause pain referred to the second branch of the fifth nerve because of the communication of the great superficial petrosal through Meckel's ganglion which is attached to the second branch.

I have made dissections of the great superficial petrosal nerve as have many other people. In Cunningham's textbook of anatomy [5] there are a good picture and description of the relations of the great superficial petrosal nerve, so that what I have to offer in anatomic studies is not at all new, and it is surprising to me that those studying pathologic

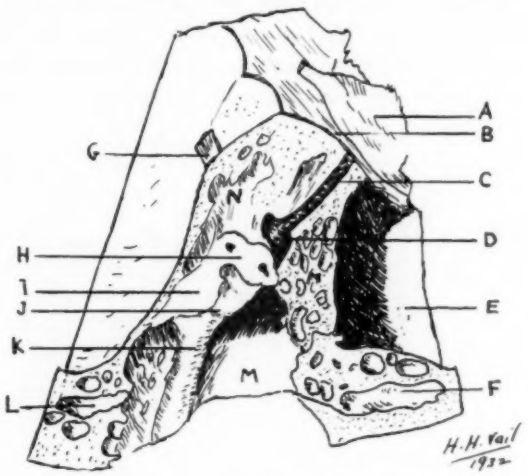

Fig. 1.—Dissection of the temporal bone viewed from above: *A*, gasserian ganglion; *B*, dura; *C*, great superficial petrosal nerve; *D*, geniculate ganglion of the facial nerve; *E*, floor of the middle fossa; *F*, zygomatic cells; *G*, facial and auditory nerve; *H*, superior vertical semicircular canal; *I*, portion of the labyrinthine capsule; *J*, eminence of the horizontal semicircular canal; *K*, ridge of the facial nerve; *L*, pneumatic cells of the mastoid; *M*, floor of the middle ear; *N*, pneumatic cells of the petrous apex.

changes in this region should not have considered more seriously the great superficial petrosal nerve.

The great superficial petrosal nerve is a fair sized nerve and is extradural throughout its entire course. It arises from the geniculate

3. Kopetzky, Samuel, and Almour, Ralph: Ann. Otol., Rhin. & Laryng. **39:** 1005 (Dec.) 1930.

4. Eagleton, W. P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20:**386 (March) 1930.

5. Cunningham, D. J.: Manual of Practical Anatomy, ed. 5, New York, William Wood & Company, p. 209.

ganglion of the facial nerve, emerges from a canal on the anterior surface of the petrous bone at a plane somewhat external to that of the internal auditory meatus, and passes downward and forward parallel to the superior border of the petrous bone in the long axis of the petrous bone to the internal carotid artery. Some dissections showed the nerve covered by bone for only a short distance; others showed it running in a tunnel, so to speak, but by the time it reached the carotid artery it was always lying free. It crosses the internal carotid artery to unite with the great deep petrosal nerve just below the lacerate foramen where the vidian canal takes its origin. As the great superficial petrosal nerve approaches the lower part of the apex of the anterior surface of the petrous apex it lies against the bone and is covered by dura to which it is only slightly adherent, while above the dura lies the gasserian ganglion in Meckel's cavity. Between the nerve and the dura below the gasserian ganglion there seems to be an increase in the amount of

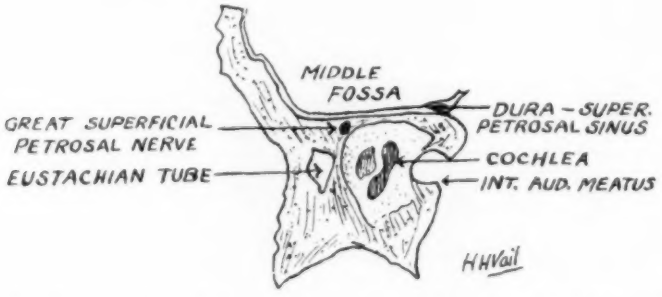

Fig. 2.—Cross-section of the temporal bone at the level of the internal auditory meatus. It shows the great superficial petrosal nerve running in a bony canal. In this particular section there are no pneumatic cells.

connective tissue, as there is a distance of from 1 to 2 mm. between the upper margin of the great superficial petrosal nerve and the lower surface of the ganglion. Thus the great superficial petrosal nerve is more closely related to the petrous bone than the gasserian ganglion or its branches.

Eckert-Möbius,[6] by means of serial sections of the petrous bone, showed the close relation microscopically between the great superficial petrosal nerve and a peritubal pneumatic cell.

The great superficial petrosal nerve unites with the great deep petrosal nerve from the carotid plexus to form the vidian nerve, which passes through the vidian canal in the sphenoid bone into the spheno-

6. Eckert-Möbius, A.: Miscroskopische Untersuchungstechnik und Histologie des Gehörorgans, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 6, p. 242.

palatine ganglion in the sphenomaxillary fossa. Among the branches from this ganglion are the orbital branches.

Vogel [7] recently worked out the branches of the sphenopalatine ganglion by means of serial microscopic sections, and stated that many nerve fibers leave the ganglion from its upper portion partly to end in the upper portion of the sphenomaxillary fossa, in the periosteum of the posterior border of the sphenoid bone and in the periorbita, and partly through the inferior orbital fissure to reach the orbit and there to communicate with branches of the first division of the trigeminus. One thus has a perfectly sound anatomic explanation of the referred ocular pains from suppuration of the petrous apex.

Suppuration in disease of the petrous apex eventually involves the gasserian ganglion and the dura about it, as has been shown repeatedly

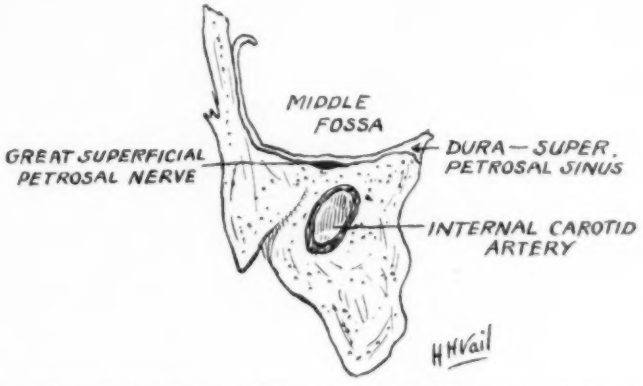

Fig. 3.—Cross-section three eighths of an inch deeper toward the midline. It shows the great superficial petrosal nerve lying under the dura.

at autopsy. That is what can be expected in the fatal cases. Yet Uffenorde,[8] in cases showing no subjective disturbances of the trigeminal tract, found on pathologic examination a severe purulent infiltration of the gasserian ganglion.

When one further considers that the ocular pains so typical of suppuration of the petrous apex are also the typical ocular pains of what Sluder [9] called sphenopalatine ganglion neuralgia and what I [10] have

7. Vogel, Klaus: Die Beziehungen des Ganglion sphenopalatinum zu den von ihm ausgehenden Nervenstämmen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:435 (March) 1930.

8. Uffenorde, W.: Eine zu vielfachen Verwicklungen führende, sehr ausgedehnte perilabyrinthäre Eiterung im histologischen Bilde, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:288, 1929.

9. Sluder, Greenfield: Etiology, Diagnosis, Prognosis and Treatment of Sphenopalatine Ganglion Neuralgia, J. A. M. A. **61**:1201 (Sept. 27) 1913.

10. Vail, H. H.: Vidian Neuralgia from Disease of the Sphenoid Sinus, Arch. Surg. **18**:1247 (April) 1929.

called vidian neuralgia, perhaps one may feel that the question I raise is not such a simple one after all. Therefore, it has been necessary to review the anatomy of the great superficial petrosal nerve and also to speak about vidian neuralgia.

Vidian neuralgia is a pain of neuralgic character in the nose, face, eye, ear, head, neck and shoulder, occurring in severe attacks. These cannot be caused by any external stimulation, are not relieved by opiates and are not associated with any subjective loss of sensation. The attacks are most often unilateral, are often nocturnal and may or may not be associated with the subjective symptoms of a nasal sinusitis. Vidian neuralgia results from an irritation of the vidian nerve by disease in the sphenoid sinus. As the vidian nerve is composed of the great superficial petrosal and the great deep petrosal, any pain from its irritation is referred throughout its distribution.

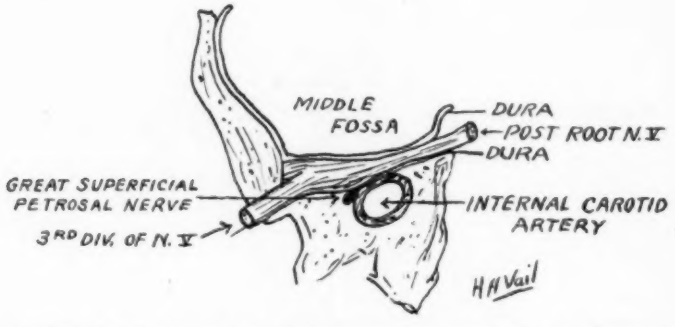

Fig. 4.—Section at the region of the gasserian ganglion. It shows the great superficial petrosal nerve lying on the carotid artery.

I have already described the pathways of referred pain from irritation of the vidian nerve. The great deep petrosal is apparently of minor importance so far as referred pain is concerned. The great bulk of the sensory elements in the vidian nerve lies in the great superficial petrosal nerve. Microscopic section of the vidian nerve allows a ready differentiation between the great superficial and great deep petrosal nerves. That the painful stimuli in cases of suppuration of the petrous apex pass backward to the geniculate as well as forward to the sphenopalatine ganglion is obvious from a study of the distribution of the pains in the head. The patients complain not only of severe pain in and back of the eye, but of deep temporal, suboccipital and auriculotemporal pain.

Now compare such subjective symptoms with three typical cases of vidian neuralgia from inflammation of the sphenoid sinus.

REPORT OF CASES

CASE 1.[11]—H. B., a man, aged 36, had severe paroxysms of pain starting on the left side of the nose just below the inner canthus, radiating into the left eyeball, forehead, upper teeth, with the exception of the incisors, palate, ear, neck and shoulder on that side. The attacks appeared at any time of the day or night, though most frequently at mid-day. An operation on the sphenoid sinus allowed the interior of the sinus to be inspected by means of the Holmes nasopharyngoscope, and a prominent ridge surrounded medially and laterally by a deep recess could be

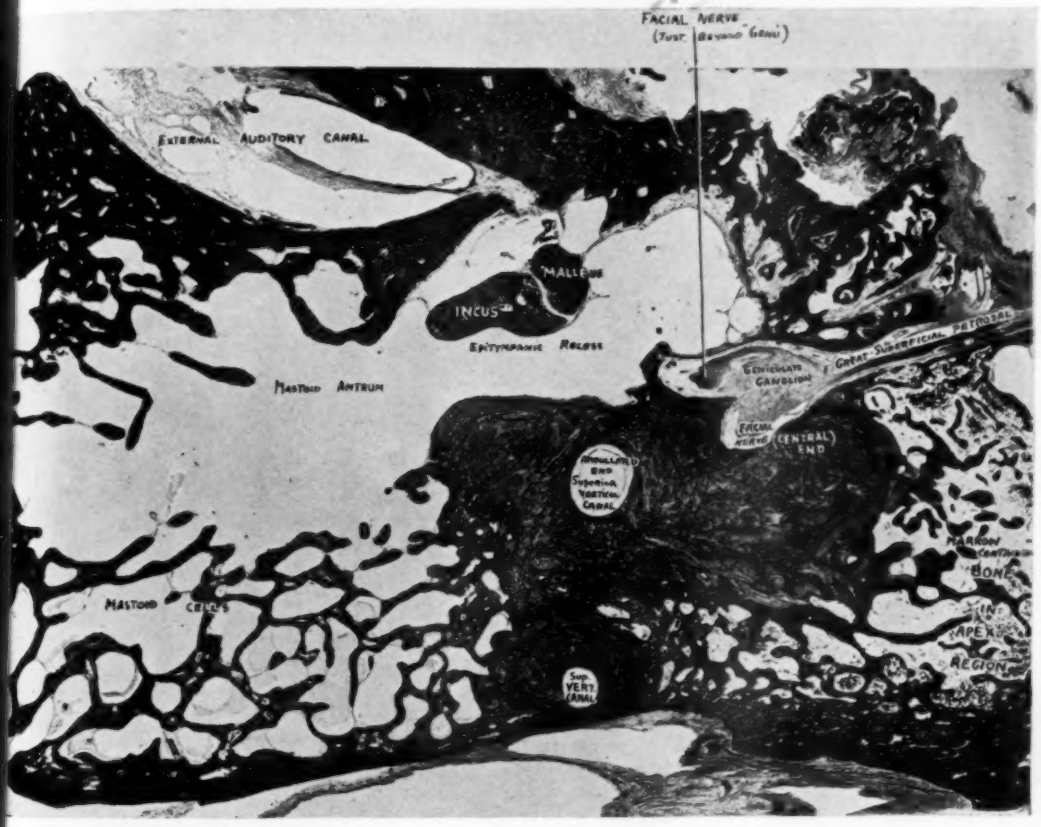

Fig. 5.—Photomicrograph of a horizontal section of the temporal bone showing the geniculate ganglion and the great superficial petrosal nerve passing from it in a bony canal, the walls of which in a few places show a dehiscence in the bone. About the great superficial petrosal nerve are pneumatic spaces.

seen. At the time of the first examination the mucosa of the sinus was a normal pale grayish color with few blood vessels, while over the ridge it was of a yellowish color.

While iodized poppy seed oil 40 per cent was being injected into the sphenoid sinus, a typical attack was induced. In the seven years that I have seen the patient there have been recurrences of the pain about once a year, and at each time severe

11. This case was reported in 1929.[10]

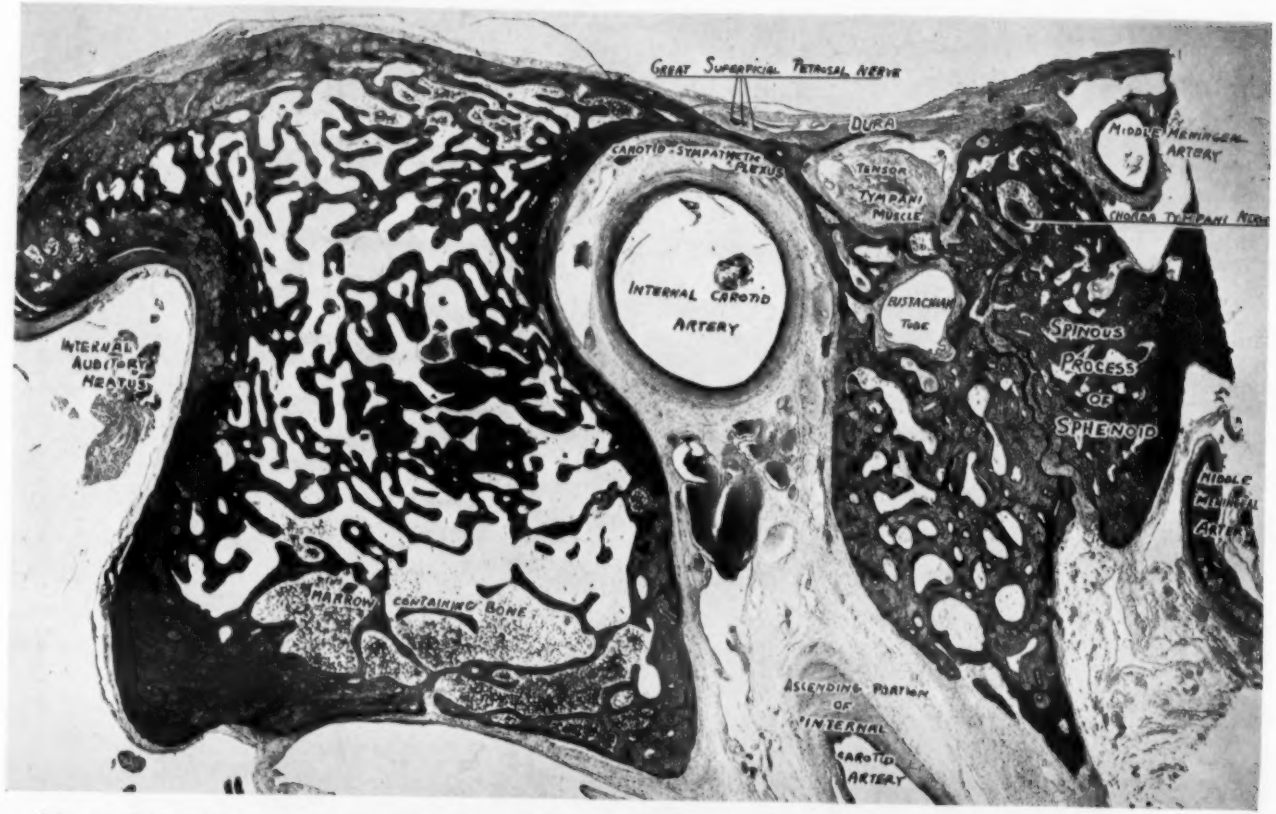

Fig. 6.—Photomicrograph of a vertical cross-section of the petrous apex. It shows the great superficial petrosal nerve lying in the dura and separated from the internal carotid artery by a thin plate of bone. The structure of the petrous apex in this section is of the diploetic type. The section shows the articulation of the petrous apex with the sphenoid bone.

inflammatory changes could be observed in the sphenoid sinus. Ten per cent cocaine injected into the sinus relieved the pain temporarily.

CASE 2.—Sister E., aged 51, was first seen on Sept. 13, 1929. She gave a history of having had a bilateral redical operation on the antrum nine years previously and a bilateral ethmoid-sphenoid operation three years before examination. On the right side for the previous year she had severe attacks of pain confined to the bridge of the nose and the back of the eyeball. It felt as though the eyeball were being pushed out. On the left side she had pain in the back of the head radiating into the back of the ear, above the ear, through the temple and into the forehead.

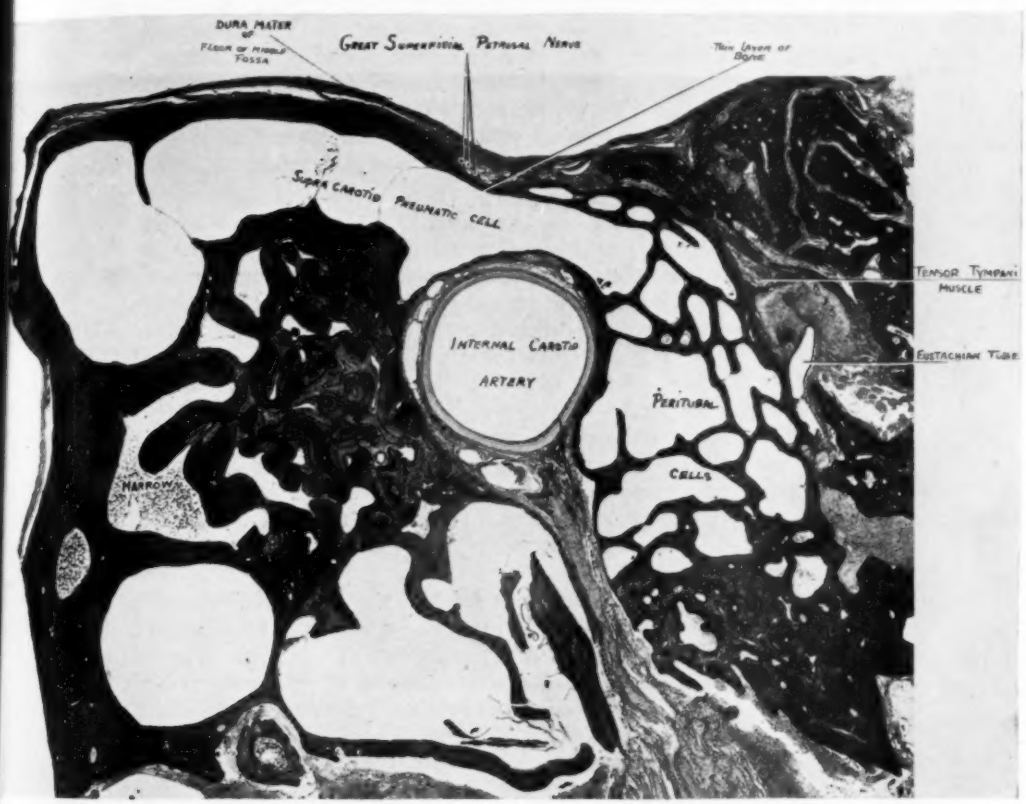

Fig. 7.—Photomicrograph of a vertical cross-section of the petrous apex where there is extensive pneumatization It shows the great superficial petrosal nerve lying under the dura of the middle fossa and to be separated by a very thin layer of bone from a large supracarotid pneumatic cell which places it in close relation to the peritubal cells. The photomicrographs which form figures 5, 6 and 7 were obtained through the courtesy of Dr. Stacy R. Guild of the Johns Hopkins Medical School of Baltimore. Dr. Guild's preparations show far better than any gross dissection the intimate relation of the great superficial petrosal nerve to pneumatic cells present in the petrous apex. It does not require any imagination to visualize the effect which suppuration in the supracarotid cell in figure 7 might have on any nerve tissue adjacent to it.

The right sphenoid opening was the size of a pin-point, and pus could be seen escaping from it. The patient readily consented to another operation on the sphenoid. The cavity of the sinus was filled with polypi and pus. I had already removed some polypi and was removing another which seemed to arise from the lateral wall of the sphenoid, when the patient suddenly complained of sharp pain in the right eye so severe as to make her jump up from the operating chair. The anesthesia was quite satisfactory, and more polypi were removed without causing any more referred ocular pain. The day after the operation there was complete relief of the pain which she said had been present daily and constantly for a year. She said that it seemed to her as if a big load had been lifted off the eye. In the fall of 1930 there was a return of the ocular pain along with a cold. This cleared up promptly with a few irrigations of the sphenoid sinus. Again in April, 1931, the pain in the right eye returned. At this time examination showed a red swollen mucosa in the right sphenoid sinus. Apparently one irrigation of the sinus relieved this pain, and I did not see the patient until nearly a year later, when she returned stating that she had been quite free from the ocular pain except for a few mild attacks with colds.

CASE 3.—Mr. G., aged 52, was first seen on Feb. 5, 1932, with a history that for six years he had had attacks of severe pain in the right eye with the feeling that the eye was being pushed out. The attacks came about once a week, most often during the night, lasted for a few hours and were relieved by hot or cold applications. Apparently there was no relation to colds. In the past year he had noticed, in addition, pain in the back of the head, always on the right side. In the past five weeks the pains had been constant in the right eye and the right side and back of the head. He had not noticed any sensory disturbances of the face. The nasal septum was badly deviated to the left from pressure by an enlarged pneumatic right middle turbinate.

An operation on the septum, middle turbinate, posterior ethmoid and right sphenoid was done under local anesthesia. The middle turbinate contained a very large turbinate cell. Polypi were present in the posterior ethmoids. The sphenoid sinus mucosa was grayish.

For the first two days after operation the pain was severe and just as bad as prior to operation, but on the third night the patient had relief from the pain, the first time for over a month. In the next few days there was great improvement, but ten days after operation he had a severe flare-up of pain, and at this time sphenoid irrigation brought out quantities of dark brown clotted blood. Daily sphenoid irrigations were done, and a month after the operation the sinus contained little pus. Following the operation the patient had only three attacks of pain. The last attack of pain was probably produced by the injection of 6 cc. of iodized oil into the right sphenoid, as at the time of this injection the mucosa was only slightly congested and two days later when the pain returned the mucosa was much congested and swollen, with pus to be seen in the cavity of the sinus.

As the inflammation in the sphenoid sinus cleared up, the pain in the eye and back of the head did likewise.

A nasopharyngoscope passed into the sphenoid sinus showed the ridge of the vidian nerve with a recess on either side. The ridge projected almost three eighths of an inch (0.9 cm.) above the floor of the sinus. The x-ray films with iodized oil injected into the sphenoid sinus show the peninsula-like outline of the vidian canal with the deep recess lateral to and below it. In this particular case the iodized oil lay below the vidian canal. Of all the cases I have seen, this showed the most extensive surrounding of the vidian canal by the sphenoid mucosa.

SUMMARY

1. Anatomic studies show the close relation between the great superficial petrosal nerve and any pneumatic cells which may be present in the anterior portion of the petrous bone.

2. It has also been shown that impulses coming over the great superficial petrosal nerve can reach the orbit and from there pass over the terminal branches of the ophthalmic division of the trigeminus by means of the anastomosis between these nerves and the orbital branches of the sphenopalatine ganglion.

3. Three cases of vidian neuralgia are reported to show that irritation of the vidian nerve in the floor of the sphenoid sinus causes ocular and orbital pain.

4. The ocular and orbital pains described by patients with suppuration of the petrous bone show a great similarity to those found in cases of vidian neuralgia.

5. The great superficial petrosal nerve is extradural throughout its course. The gasserian ganglion and its divisions are above the dura. Hence, there must be a dural involvement before the gasserian ganglion and its branches are affected.

CONCLUSION

It would seem that the ocular and orbital pains so typical of petrous suppuration in the early stages are not due to an irritation of the ophthalmic division of the fifth nerve, but are produced by the irritation of the great superficial petrosal nerve caused by the suppuration in the petrous bone.

ENDONASAL RADICAL OPERATION ON THE ANTRUM

ALFRED WACHSBERGER, M.D.
NEW YORK

The object of this paper is to stress a method of approach in the performance of a radical operation on the maxillary antrum which, although not new, can in most cases be advantageously used in preference to the operative procedure usually employed. It has not been generally accepted as yet to the extent which its merit warrants.

Until 1908 the usual treatment for acute and chronic empyema consisted of drainage through the mouth, using either the dental alveola or an opening into the facial wall above the gingiva. Drainage through the nose was advocated by Mikulicz [1] and Krause,[2] but was not generally practiced because of the belief that successful drainage had to be instituted at the lowest point of the antrum. Unfortunately, these openings rarely closed permanently, with the result that pus drained into the mouth and food lodged in the antrum, unless this was prevented by using an obturator. These inconveniences and the fact that better results could be obtained by intranasal drainage popularized the conservative methods of treatment which are in use today.

Radical procedures, especially those advocated by Caldwell-Luc and Denker, are major operations requiring many days of hospitalization, and the percentage of favorable results is not in proportion to the extent and risk of the procedure. To overcome these and other disadvantages, Sturman [3] and, three months later, Canfield [4] introduced independent of each other, in 1908, a method of operation which is the subject of this communication.

Grossly, the difference between this procedure and the Caldwell-Luc or the Denker operation is that the oral incision is eliminated and the operation is performed entirely through the nostril. This is its major difference from the Denker operation, to which it otherwise conforms in principle. It is a radical procedure and must not be classed with

1. Mikulicz: Arch. f. klin. Chir. **34**:626, 1886.

2. Krause, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen-, Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 2, p. 736.

3. Sturman: Zur Behandlung der Oberkieferhoehleneiterungen, Berl. klin. Wchnschr. **39**:684, 1902; Die intranasale Eroeffnung der Kieferhoehle, ibid. **45**: 1273, 1908; Erfahrungen mit meiner intranasalen Freilegung der Kieferhoehle, Arch. f. Laryng. u. Rhin. **48**:143, 1910.

4. Canfield, R. Bishop: The Submucous Resection of the Lateral Nasal Wall in Chronic Empyema of the Antrum, Ethmoid and Sphenoid, J. A. M. A. **51**:1136 (Oct. 3) 1908.

the usual endonasal window operation under the inferior turbinate for opening into the maxillary antrum.

The Canfield-Sturman operation is indicated whenever a radical operation on the maxillary sinus is warranted. There are a few exceptions in which the combined oral and nasal approach becomes preferable. These include (1) cases in which there is a tumor formation, particularly a large tumor of suspected malignancy, and (2) cases of dental origin in which there is involvement of the alveolar process of the upper maxilla, like osteomyelitis occurring after the extraction of a tooth.

TECHNIC OF OPERATION

When the operation is performed under local anesthesia, the patient receives a hypodermic injection of scopolamine hydrobromide, from one one-hundredth to one two-hundredth grain (0.6 to 1.3 mg.), combined with morphine, from one-eighth to one-fourth grain (8.1 to 16.2 mg.), one hour before operation. It is important that the patient be kept in a darkened room and in a reclining position from the time of injection to the time of operation. The operation is performed preferably with the patient in a semirecumbent position. Halle operates with the patient sitting up. The vibrissae are first cut short because they disturb the operator's vision and also to secure better sterility. Four per cent tincture of iodine, followed by alcohol, is swabbed over the tip of the nose, the upper lip and the nasal vestibule.

For local anesthesia, both topical application and conduction anesthesia are employed. Three cotton plugs, soaked in 10 per cent or 30 per cent alypin and epinephrine, equal parts, and squeezed to remove the excess fluid, are inserted into the nasal cavity: one into the middle meatus, one between the septum and the inferior turbinate and the third between the inferior turbinate and the naso-antral wall. Where this space is too narrow for insertion of the plug, careful epinephrinization will widen it sufficiently for the purpose. The cotton is left in place for fifteen minutes. For blockage of the sphenopalatine ganglion, I use procaine hydrochloride, 0.5 per cent, injected by the external buccal and infrazygomatic route. On withdrawing the cannula, I deposit some procaine hydrochloride at the tuber maxillare in order to anesthetize the posterior superior dental nerves which enter the antrum through narrow canals in the posterior wall. This procedure saves anesthetization of the antral lining after the sinus is opened. After the cotton plugs are removed from the nasal cavity, procaine hydrochloride is injected subperiosteally into the naso-antral wall and the floor of the nose. An injection is also made into the ala, the tip of the nose and the cutaneous septum in order to prevent pain on stretching the nostril with the speculum. The soft parts of the facial antral wall should be well infiltrated—for the purpose of securing easy elevation and anemization—by leading the cannula from the nasal vestibule between the pyriform crest and the ala along the facial wall of the antrum as close to the bone as possible in the direction of the infra-orbital foramen. Thus a complete anesthesia of the operative region is obtained.

In younger and in nervous persons a general anesthesia, preferably by the intratracheal method, may be employed. It is not preferable to a local anesthesia because of the greater amount of bleeding which results even when it is combined with a local infiltration. Furthermore, there is the risk from ether anesthesia in

operations on the head in general. Whenever possible the operation should be done under local anesthesia.

The incision is made in the nasal vestibule in the fold which forms between the pyriform crest and the ala when the speculum is inserted and spread (fig. 1 *A*). In order to get on the buccal side of the crista, the scalpel must be held parallel to the sagittal plane of the patient. The incision starts at the level of the insertion of the inferior turbinate, and is drawn downward and parallel to the pyriform crest to the level of the nasal floor. Care must be taken that the periosteum is severed with this incision. The bleeding occasionally encountered from the angular artery can usually be stopped by the stretching of the edges of the wound with the speculum and rarely requires ligation. I never had to resort to it in my cases.

For separation of the soft parts of the cheek from the bony facial antral wall, Joseph's elevator is advantageously used because it is rigid, curved in the plane and sharp. The separation should not be extended to expose the infra-orbital

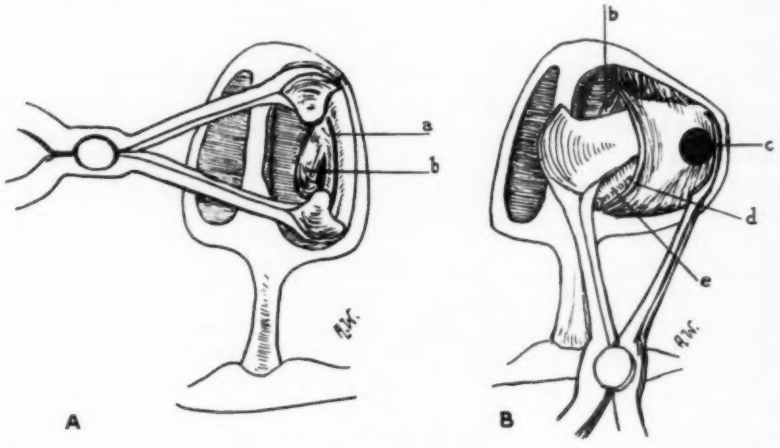

Fig. 1.—*A*, incision along the crista pyriformis; *B*, the exposure of the bony facial and naso-antral wall and the trephine opening into the antral cavity: *a*, incision; *b*, inferior turbinate; *c*, trephine opening; *d*, crista pyriformis, and *e*, mucoperiosteum naso-antral wall.

nerve at its exit, lest there result a period of severe postoperative pain. In order to separate the mucoperiosteum from the naso-antral wall, the periosteum is freed from the pyriform crest, where it is found closely adherent. This is done by scratching the fibers off with the edge of a chisel. The rest of the elevation is easily performed by closely hugging the bone. Sturman, according to his original description, separated beyond the insertion of the inferior turbinate. He fractured the attachment, but left the turbinate in contact with the mucoperiosteum. He then elevated the mucosa in the middle meatus. I have not found this necessary to secure a sufficient exposure, and limit my separation to the extent of the inferior meatus, stopping at the insertion of the inferior turbinate. A medium-sized Killian speculum or a special speculum with one long and one short branch is now inserted into the wound, exposing the medial and outer walls of the antrum. The two branches of the speculum protect, on their outer side, the soft tissues of the cheek and the mucoperiosteum with the inferior turbinate, respectively.

By the spread of the speculum the inferior turbinate may be infracted medially, if necessary, for better exposure. The antral walls now appear between the branches of the speculum, not unlike the bony nasal septum in a submucous resection (fig. 2).

The antrum is now opened, using Halle's[5] largest-sized (8 mm.) circular trephine, which is connected to the pistol handle and driven by a motor. The trephine should be placed firmly against the facial wall at a point as far lateral from the pyriform crest as possible and at the level of the midsection of the inferior meatus. It is important to choose this place for trephining because (1) the facial wall is thinnest here and (2) it permits entrance into the antral cavity, no matter how small this may be, owing either to a standstill in the course of development or to a sclerosis of its walls. In one of my cases I encountered an antrum, the size of a large pea, situated posteriorly and superiorly

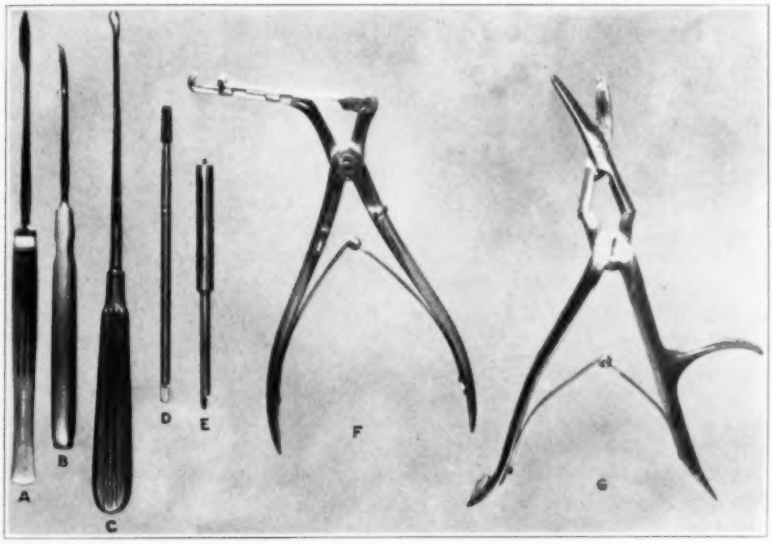

Fig. 2.—Some of the instruments used for the operation: *a*, Halle's ethmoid knife; *b*, Joseph's periosteal elevator; *c*, Ritter's curet; *d*, Halle's conical burr; *e*, Halle's trephine; *f*, Citelli's bone rongeur; *g*, the author's bone scissors.

to what would have been a normally developed sinus in the upper maxilla. When the opening is made too near to the crista pyriformis, one is liable to enter the nasal cavity and miss the antral cavity; if it is made too low, the alveolar process and the dental roots may be trephined and thus injured. With the trephine used as described, a circular opening is obtained in a few seconds (fig. 1 *B*); through which the antrum may easily be inspected for any exudate which may be present and for determining the character of the mucosa. A gouge may be used to make this opening instead of the trephine when the trephine is unavailable. The choice of

5. Halle, M.: Zur Behandlung des Empyems der Highmorshoehle, Berl. klin. Wchnschr. **37**:772, 1900. Die intranasalen Operationen bei eitrigen Erkrankungen der Nebenhoehlen der Nase, Arch. f. Laryng. u. Rhin. **29**:73, 1914.

instruments is immaterial. In case the sphenopalatine ganglion has not been blocked previous to operation, the mucosa which presents itself inside the opening may now be swabbed with a local anesthetic. When one has estimated and evaluated the pathologic changes which present themselves, the opening is enlarged by removing the facial antral wall medial to the original opening, the crista pyriformis and the bony naso-antral wall, just as is done in the Denker operation (fig. 2). This is performed by means of either the electric trephine, the saw, the chisel and mallet, Citelli's bone rongeur or the combined use of all these means. For removal of the naso-antral wall I use a special scissors made to my design by Pfau's Instrument Company. It is a modification of the Jansen-Middleton septum instrument. The blades are made on the same principle as those in a poultry shears; they are curved and have corrugated edges which prevent slipping while cutting the bone. When all the naso-antral wall lateral to the inferior meatus has been removed, there usually remains a threshold-like crest between the

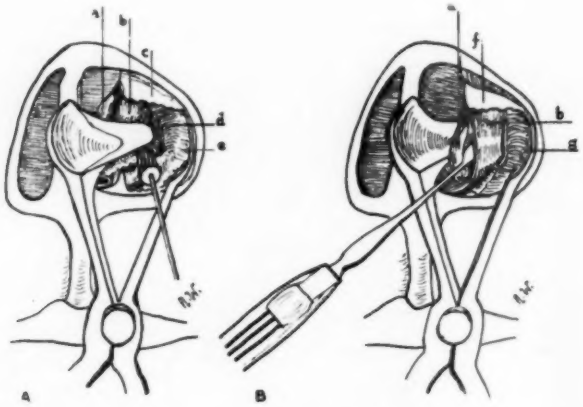

Fig. 3.—*A*, threshold between the antral and the nasal floor is leveled with Halle's conical burr. *B*, bony naso-antral wall is removed and a flap cut from the mucoperiosteum of the naso-antral wall on the nasal side: *a*, inferior turbinate; *b*, mucoperiosteum, naso-antral wall; *c*, facial-antral wall; *d*, naso-antral wall; *e*, antrum; *f*, facial wall, and *g*, antral cavity.

antral and the nasal floor. This eminence is of solid bone and must be removed entirely to establish free drainage and also to permit adaptation of the flap, which is subsequently turned into the antrum. This is done easily, quickly and efficiently with Halle's conical burr (fig. 3*A*). The burr should be used at frequent intervals rather than for too long a period at a time so as to avoid overheating and resulting bone necrosis. In the intervals the burr is dipped into sterile liquid paraffin. Posteriorly the naso-antral wall need not be removed as thoroughly as on the bottom: first, because it is not essential for adequate drainage, and second, because of the danger of injury to the sphenopalatine artery, the bleeding from which is exceedingly hard to control because of its size and location in the bony sphenopalatine canal.

With the facial and naso-antral wall removed, the interior of the antrum is sufficiently exposed for thorough inspection and curettage. With proper adjustment of the speculum and the patient's head, the posterior and lateral wall, the

floor and the orbital plate become plainly visible. The remaining part of the facial wall and the nasolacrimal recess may be inspected with the use of a postnasal mirror. All parts of the mucosa are easily reached with the curet and removed where necessary. Whether to remove the entire lining in cases in which there is only a part of it macroscopically involved or whether to leave some part in situ to be permitted to become normal by the establishment of aeration and drainage is a question of surgical judgment acquired by experience and does not fall within the scope of this paper. It is of interest here to know that every part of the mucosa can be removed as effectively by the method that I describe as by the oral route.

The final stage of the operation, the cutting of the flap, is simple (fig. 3*B*). A knife with a long stem and a short blade (Halle's ethmoid knife is preferable because of its slenderness) is introduced between the inferior turbinate and mucoperiosteum. With one incision from behind forward along the attachment of the turbinate, a good-sized flap is cut, which will fall naturally and cover the antral floor. It is advisable to thin the anterior edge of the flap so that it may not form an eminence and obstruct the view into the interior of the antrum during after-treatment.

The anterior tip of the inferior turbinate need not be removed because, when the pyriform crest is removed, the opening to the antrum lies in front of and to the lateral side of the turbinate and it can easily be entered with a straight cannula when necessary for postoperative suction and irrigation. No packing is used. In cases in which part of the antral lining is left in place, more than the usual amount of bleeding may be encountered. A finger-cot stuffed with gauze packing introduced into the antrum will control such bleeding. This dressing is removed on the day following operation.

Postoperative treatment is simple. The patient is hospitalized for two or three days. Sturman sent his patients home after operation and had them return for after-treatment. Some swelling of the cheek usually occurs on the second day; yet this is infinitely less than after the Caldwell-Luc or Denker operation. I do not use irrigation because I believe that a thorough removal of the wound secretion retards the regeneration of the lining. It is better to employ gentle suction with a straight cannula instead, but not before about the fifth day. The regeneration of the mucosa can be observed visually. This is possible even with the inferior turbinate intact because of the removal of the pyriform crest. This direct view into the antrum is extremely helpful when excessive granulations have to be removed. The opening will become slightly narrower during the postoperative course. Then epinephrinization of the inferior turbinate or the use of a nasopharyngoscope may be helpful for thorough inspection of the cavity. I have never seen the opening close completely, even after years of reexamination. The usual length of time for postoperative treatment is from three to four weeks. If the operation is performed during an acute exacerbation of the chronic condition, a longer period of time may be required for a cure. Cases which show excessive formation of granulations or cystic degeneration of the newly formed lining should lead the surgeon to suspect drainage of pathologic secretion from infected ethmoid cells which irritates the newly forming antral mucosa. I believe that whenever there are indications for a radical antrum operation, the adjacent ethmoid cells will usually present pathologic changes, which are either primary or secondary to the chronic antral infection. These cells must be inspected and, when necessary, curetted during the operation.

Halle [6] has recently described an endonasal operation for the antrum which he terms a radical procedure even though it is still more conservative than the method described. It is based on his operation for ozena, which leaves the facial wall and pyriform crest intact and confines itself to the removal of the naso-antral wall. The reason for Halle's conservatism in the removal of the facial wall is to save the nervi alveolares superiores, anteriores and mediales which, when injured, may cause the well known paresthesias of the teeth after the operation. The exposure which is necessary for visualization and instrumentation of the interior of the antrum is achieved by means of a vertical incision which begins at the midsection of the middle meatus and ends at the nasal floor. This incision lies anterior to the head of the inferior turbinate and posterior to the crista pyriformis. The naso-antral wall is fractured along this incision and pushed toward the septum by the spread of the inserted speculum.

It is my experience with this approach to the antrum that although the exposure is not as complete as in the Canfield-Sturman method, it is applicable to patients with large antrums rather than to patients with a sclerosis of the antral walls, which manifests itself most markedly in the pyriform crest; the latter then protrudes over the concave surface of the naso-antral wall and bars the view into the antrum. Paresthesia of the teeth from injury to the dental nerves while removing the facial wall is usually of transient quality. Real damage to the dental pulp arises more often from accidental uncapping of the roots while curetting the antral floor.

The Canfield-Sturman antrum operation is a procedure which permits visibility and accessibility of every part of the mucous lining and allows surgical intervention in all its parts. A comparison between this and the methods of Caldwell-Luc and Denker follows.

The elimination of the oral incision presents distinct advantages to the patient as well as to the surgeon. No bucco-antral fistula can develop through which the stratified squamous epithelium of the oral cavity may grow into the antrum, thus delaying healing and establishing a nonphysiologic condition in the antrum. Lodging of food in the antrum and inability of the patient to blow the nose properly are prevented. The necessity for secondary plastic procedures to close a pathologic fistula is obviated. The time of operation is shortened and the surgical procedure minimized in the eyes of the patient. The inferior turbinate is kept intact, and it does not interfere with the inspection of the cavity; rather, inspection is facilitated by the removal of the pyriform crest. This also allows direct ventilation by the inhaled air, which is beneficial

6. Halle, M.: Schaedigung der Zaehne nach Radikaloperation der Oberkieferhoehle, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **126**:251, 1930.

for the promotion of healing. The operative technic is rather simple and, comparatively speaking, no more difficult than a submucous resection of the septum as far as its technic is concerned.

Using this procedure, I have operated on over fifty patients in Halle's Clinic in Berlin and, during the last few years, on thirty-two patients at the Polyclinic Hospital and Beth Israel Hospital in New York. Because of the gratifying results of the technic described, I advocate this operation as the procedure of choice when a radical operation on the maxillary antrum is to be undertaken.

51 West Seventy-Third Street.

ACUTE LARYNGOTRACHEOBRONCHITIS IN INFANTS

REPORT OF THREE CASES

MALCOLM C. JOHNSON, B.S., M.D.
FORT SMITH, ARK.

Acute laryngotracheobronchitis causing dyspnea in infants may occur in the course of an acute infection of the upper respiratory tract, or be secondary to the presence of a foreign body in the trachea or bronchi. As a rule, the history gives sufficient evidence as to which factor is the cause, but occasionally cases are seen in which the differentiation is not easily made. In the past year I have seen three cases in which the history gave no clue or was misleading.

Most papers on the subject of this disease refer to it as a rare condition, and state that the references in the literature are few. Gittins,[1] however, has been able to find thirty-one references to this disease. Included in this list is the article by Baum,[2] which is most complete.

CHARACTERISTICS OF THE CONDITION

Etiology.—Acute laryngotracheobronchitis is, as a general rule, preceded by a so-called cold with symptoms of nasal discharge, cough and elevation of temperature. However, it may be primary in the larynx or trachea. This condition may also occur following the aspiration of a foreign body into the trachea or bronchi. This is most apt to occur with vegetative bodies. It is primarily a disease of children. Various organisms have been found in smears and cultures taken from the larynx and trachea in these cases, namely, staphylococci, streptococci, pneumococci and the influenza bacillus. As a rule, a mixed infection is present. In the infective forms it has appeared in epidemics.

Pathology.—The pathologic observations consist of pronounced inflammation and swelling of the subglottic area, accompanied by redness of the tracheal mucosa below the swollen area. The arytenoids usually show a slight congestion. The true cords may show no departure from the normal, or they may be slightly congested. One of the most

1. Gittins, T. R.: Laryngitis and Tracheobronchitis in Children; Special Reference to Non-Diphtheritic Infection, Ann. Otol., Rhin. & Laryng. **41**:422 (June) 1932.

2. Baum, H. L.: Acute Laryngotracheobronchitis, J. A. M. A. **91**:1097 (Oct. 13) 1928.

characteristic observations is the presence of thick, heavy, tenacious mucus in the trachea. Plugs may be formed by the drying of this secretion. The swelling produced by the subglottic edema presents a marked obstruction to the passage of air.

Symptoms.—Dyspnea is the most striking symptom of this disease. In the infectious cases it is preceded by an acute infection of the upper respiratory tract. As a rule, a dry, croupy cough accompanied by fever of a moderate degree is one of the early symptoms. The absence of fever may be misleading. Baum states that late in the disease, owing to insufficient oxygenation of the blood, fever may be absent, but that it recurs after a free passage of air has been established. As dyspnea increases in severity, suprasternal, supraclavicular and epigastric retraction appear. The pallor that precedes cyanosis then makes its appearance. The heart rate is rapid, owing to the insufficient supply of oxygen and also to the extreme effort necessary to breathe through a constricted passage. The late picture is one of approaching death.

Findings.—Direct examination may show some injection of the epiglottis, the ventricular bands and the cords. The striking feature is the intense redness and swelling of the subglottic region. The tracheal mucosa is red and velvety. A pseudomembrane may be present. When the trachea is opened, a thick mucoid secretion is encountered. Plugs formed by the drying of this material are likely to be found.

Examination of the chest may reveal the presence of bronchopneumonia or lobar pneumonia with the usual findings. The failure of air to enter one of the lungs owing to occlusion of one of the bronchi by a dried mucus plug may be noted in some cases.

DIAGNOSIS AND TREATMENT

Diagnosis.—Acute laryngotracheobronchitis has frequently been mistaken for laryngeal diphtheria. This is unfortunate, since dependence has been placed on the use of antitoxin alone as the method of treatment. One point of distinction lies in the fact that the changes in the nondiphtheritic disease are, as a rule, limited to the subglottic region. However, smears and cultures should be taken in all such cases. In most of the cases reported, antitoxin has been given, the author stating that if the case was not one of diphtheria, the reaction produced by the foreign protein could be only of a beneficial nature.

The question as to whether the condition is of an acute infectious nature or secondary to the presence of a foreign body sounds relatively simple. However, I have not found the distinction so easy to make in some cases. It would seem that the presence of fever and a history of a cold would render the diagnosis of the infectious type relatively easy. However, these factors are not present in all cases. In the type of case

in which the condition is primary in the larynx and trachea, the question is still more difficult to answer. Roentgen examination in the hands of an expert is of great value in making the diagnosis of nonopaque foreign bodies, but the interpretation is not so easily made by the average roentgenologist. Frequently the initial symptoms of coughing and choking following the aspiration of a foreign body may pass unnoticed. This is especially true in the case of an infant who is not under close supervision.

Treatment.—Two methods of treatment for this condition have been offered, namely, intubation and tracheotomy. Baum feels that intubation should be given a trial and later be followed by tracheotomy if necessary. Gittins believes that tracheotomy is the treatment of choice. I believe this method to be best, as it also offers an opportunity to search the trachea and bronchi for the presence of a foreign body in cases in which the causative factor is questionable. Also, it gives a better chance to remove the thick mucus and any plugs present. Intubation should be done only in cases in which the child is in the type of hospital where, if the tube is expelled, it can quickly be replaced. Obviously, in a general hospital tracheotomy is much safer than intubation.

In all of my cases it was necessary to use suction through the trachea frequently. The formation of thick, heavy, tenacious mucus which the child was not able to expel through the tube rendered this necessary. Bronchoscopic removal of dried mucus plugs from the trachea is frequently required. The tracheal tube may have to be left in place for as long as three weeks or more in some cases. Inhalations of steam with the administration of sodium bicarbonate have been recommended as a means of keeping the secretion more liquid. Diphtheria antitoxin is given as a precautionary measure.

Prognosis.—These cases should be given a guarded prognosis. Many reports give a 50 per cent mortality, pneumonia being the cause of death in practically every case.

REPORT OF CASES

The following three cases are examples of the type of case in which the absence of fever and a preceding cold caused doubt as to whether the case was one of an infectious nature or was secondary to the aspiration of a vegetative foreign body.

CASE 1.—H. L., a boy, aged 11 months, was brought to the hospital showing marked difficulty in breathing. Suprasternal, supraclavicular and epigastric retraction were marked. The child was pale and restless. Dyspnea had been present for the past twenty-four hours, and had become progressively worse. The mother stated that she thought the child might have had a slight cold a few days previously. No history of choking or gagging could be elicited. The temperature was 98 F.;

the pulse rate, 110, and the respiratory rate, 40. Direct examination revealed a slight redness of the arytenoids and the ventricular bands. The cords were slightly inflamed. The subglottic region was swollen practically shut. Passage of a 4 mm. bronchoscope was attempted, but discontinued because of the intense swelling. A no. 1 intubation tube was inserted under direct vision. Shortly afterward the temperature rose to 105 F. Smear and culture showed staphylococci and streptococci mixed. No diphtheria bacilli were found, but 10,000 units of antitoxin were given, notwithstanding the negative report. Roentgenograms were negative as regards a foreign body. The tube was removed on the fifth and twelfth days, but had to be reinserted immediately on each occasion. Owing to financial conditions, the patient was removed from the hospital. Tracheotomy at this time was advised, but refused. Three days after the child returned home, the tube was coughed up, and an emergency tracheotomy was necessary. The child was then returned to the hospital. It was necessary to use suction through the tracheal tube on numerous occasions to remove the thick mucus present and to allow the child to breath freely. Bronchoscopic examinations through the tracheal wound were made on several occasions. Dried mucus plugs were found and removed, but no evidence of a foreign body was found. The tracheotomy tube was removed on the twelfth day, and the child's convalescence was uneventful.

Case 2.—C. H., a boy, aged 2 years, entered the hospital markedly cyanosed. Dyspnea also was marked. Suprasternal, supraclavicular and epigastric retraction were present. The parents stated that three weeks previously the child had a slight cough which became progressively worse, and that dyspnea had been present for the past week. A history of choking or gagging could not be elicited. The temperature on admission was 97.8 F., the pulse rate, 128, and the respiratory rate, 42. Examination of the chest did not reveal any areas of dulness, and breath sounds were heard over both lungs. Direct examination showed the cords and the arytenoids normal. Marked swelling of the subglottic region was present. Respirations ceased during this examination. A tracheotomy was performed, and respirations were resumed. When the trachea was opened, a large, thick, heavy, yellow mucus plug was expelled through the incision. The axillary temperature rose to 101.2 F. following tracheotomy. It was necessary to use suction through the tracheal wound several times daily for the first few days, owing to the obstruction of the trachea by thick mucus. Bronchoscopic examinations made at intervals during the patient's stay in the hospital did not show the presence of a foreign body. The tube was removed at the end of twelve days.

Case 3.—E. K., a girl, aged 3 years, was brought to the hospital markedly dyspneic; the dyspnea was accompanied by retraction of the suprasternal and supraclavicular spaces. According to the history, the child had been well until the day of admission, when she choked while eating corn. It was believed that a foreign body had been aspirated at that time. The temperature was 99 F. Direct examination showed the cords and arytenoids slightly injected. The subglottic area was fiery red and considerably swollen. Tracheotomy was performed, and when the trachea was opened a thick, brownish-red piece of mucus was expelled from the incision. Suction was applied, and more of this material was aspirated from the trachea. Bronchoscopic examination through the incision showed the tracheal mucosa deeply injected Examination of the bronchial tree did not show the presence of a foreign body. After the tracheotomy, the temperature rapidly rose to 103 F. In this case considerable effort was required to maintain a free passage for air to enter the lungs in sufficient amounts. Bronchoscopic examinations were made at intervals, first, to remove the crusts of dried mucus present,

and, secondly, to attempt to find the piece of corn if present. Roentgen examination did not show anything that might be interpreted as a change due to the presence of a nonopaque foreign body. The tube was removed in twelve days, and convalescence was uneventful.

COMMENT

In cases 1 and 2, the history given and the absence of fever made the diagnosis of the infectious type of acute laryngotracheobronchitis questionable. In case 3 the history was misleading. It was believed at the time of examination that a foreign body was present in the trachea or bronchi. However, it does not seem reasonable to suppose that all the thick mucus present when the trachea was opened could form in such a relatively short space of time if its presence had been due to the aspiration of a piece of corn. Repeated endoscopic examinations made while the patient was in the hospital failed to reveal the presence of a foreign body. Since its discharge from the hospital the child has not shown thoracic symptoms on repeated examinations.

CONCLUSIONS

1. In the absence of fever and a preceding cold, the question as to the etiologic factor producing acute laryngotracheobronchitis is sometimes difficult.

2. Tracheotomy is the treatment of choice.

3. Frequent aspirations through the tracheal tube or wound are important in the treatment of this condition, as is the bronchoscopic removal of dried mucus plugs, if present.

OTITIS MEDIA IN SCARLET FEVER

HORACE J. WILLIAMS, M.D.
PHILADELPHIA

The object of this writing is to present and to discuss the aural complications in patients with scarlet fever occurring during the years from 1922 to 1929, inclusive, at the Philadelphia Hospital for Contagious Diseases. The discussion will have reference to the recorded literature on the general subject of otitis media in scarlet fever.

It seems essential to the just evaluation of the following data to understand at the outset that the Philadelphia Hospital for Contagious Diseases is a municipally owned institution, and that the overwhelming bulk of the patients admitted there are received from the less well-to-do and laboring class of people. The patients, therefore, on admission often have suffered from inadequate medical and hygienic care and improper, if not insufficient, nourishment.

During these eight years, 14,733 patients suffering from scarlet fever were admitted to the hospital. In 1,535, or 10.8 per cent, suppurative otitis media developed. This percentage compares favorably with figures from other sources. Ross [1] has collected an extensive list of patients, showing the incidence of otitis media in scarlet fever in "many epidemics in many parts of the world, during a period of many years, and under the observations of many different physicians." Table 1 is a copy of his table, with the addition of his own series of patients, a series by Strachan [2] and one by Lefler.[3]

It will be noted at a glance that the mean average is about 15 per cent. In 167, or 10.8 per cent, of the 1,535 patients with otitis media, mastoiditis requiring simple mastoidectomy developed. This number may be compared with Strachan's [2] 98 patients with otitis media in scarlet fever, in 10 of whom, or 10.2 per cent, acute mastoiditis requiring surgical treatment developed, and with Gardiner's [4] 300 patients, in 21

1. Ross, E. L.: Otitis Media in Scarlet Fever, Ann. Otol., Rhin. & Laryng. **33**:1319, 1924.

2. Strachan, J. G.: Ear, Nose and Throat Complications of Scarlet Fever, Canad. M. A. J. **14**:1089, 1924.

3. Lefler, A. B.: Scarlet Fever Otitis: A Report of Sixty-Six Cases, Ann. Otol., Rhin. & Laryng. **36**:495 (June) 1927.

4. Gardiner, W. T.: Scarlet Fever Otitis: Report on Two Years' Work at the Edinburgh City Hospital for Infectious Diseases, J. Laryng. & Otol. **37**:497, 1922.

of whom, or 7 per cent, mastoiditis developed requiring surgical treatment.

The incidence of mastoiditis in the total number of patients with scarlet fever was 1.1 per cent.

Of the 167 patients with mastoiditis, in 157 the condition was unilateral and in 10 it was bilateral. There were 18 deaths among the patients operated on for mastoiditis. This gives a percentage of 10.8. The mortality rate for the 1,535 patients with otitis media was 4 per cent.

The average number of days from operation to recovery was forty-seven and eighth-tenths in 148 operations in which the time of recovery was recorded.

Among the 1,535 patients there were 2,186 suppurating ears. Roughly, one third of the patients had a bilateral condition. Myrin-

TABLE 1.—*Incidence of Otitis Media in Scarlet Fever*

Authors	Number of Patients with Scarlet Fever	Number of Patients with Otitis Media	Percentage of Otitis Media in Scarlet Fever
Le march'hadour	339	36	10.65
Yearsley	17,829	2,355	13.20
Sprague-Bezold	17,087	64	3.75
Fowler-Bader	51	13	25.00
Fiedert	25,882	3,833	14.80
Biedert-Finley	4,397	439	10.00
Biedert-Craiger	1,008	131	13.00
Nager	744	119	16.00
Borden	746	65	8.70
Hombren	9,590	2,020	21.00
Alderton	1,621	178	11.00
McCrae	850	125	14.70
Ross	2,595	.....	14.00
Strachan	589	98	16.60
Lefler	473	58	12.25

gotomy was performed on 858 ears before the tympanic membrane ruptured, and on 233 after it had ruptured; in 1,095 ears there were ruptured tympanic membranes that were not incised. The type of myringotomy employed was free linear incision, yet the records show that in a considerable number of instances incision was repeated once or more. The incidence of mastoiditis requiring surgical treatment in these three different groups did not vary widely, being 6.6 per cent in that in which the tympanic membranes were incised before rupture; 7.7 per cent in that in which the membranes were incised after rupture, and 9.3 per cent in that in which the membranes ruptured and were not incised.

From these figures the conclusion may be drawn that early incision of a bulging tympanic membrane before it ruptures and incision of a ruptured membrane when the rupture is inadequate for proper drainage tend to lower the incidence of acute mastoiditis requiring surgical intervention (table 2).

In regard to the repeated incision of the tympanic membrane, I have come to the conclusion that it is seldom of value, provided the original incision was free. The bulging of the tympanic membrane that invited repeated incision was often due to swollen mucosa of the middle ear caused by irritation of a small amount of pus from the mastoid, which was not sufficient in amount to keep the incision open. In regard to the incision of a tympanic membrane that has already ruptured but in which the opening is inadequate for proper drainage, it is my opinion that it is of distinct value and a good practice.

It is interesting to note the incidence of otitis media according to age groups. Sixty-one and seven-tenths per cent of the patients were 5

TABLE 2.—*Incidence of Acute Mastoiditis Requiring Surgical Treatment*

Ears		Incisions				Mastoidectomies		
		1	2	3	4	Unilateral	Bilateral	Deaths
Incised	858	702	102	32	8	50	7	7
Ruptured and incised	233	183	32	14	3	17	1	4
Ruptured	1,095	...	...	..	..	90	12	7
Number of ears								2,186.0
Average number of days from operation to recovery								47.8
Average number of mastoids								148.0

TABLE 3.—*Age Incidence*

Age	Number of Patients	Percentage
0- 5	948	61.7
5-10	463	30.2
10-15	64	4.2
15-20	32	2.1
20 and over	28	1.8
Total number of patients	1,533	

years old or younger; 31.2 per cent were between 5 and 10 years; 4.2 per cent were between 10 and 15; 2.1 per cent were between 15 and 20, and 1.8 per cent were over 20 (table 3).

Harries and Gilhespy [5] reported the cases of a series of 290 patients with scarlatinal otitis, in which 40.3 per cent were under 5 years of age; 54.5 per cent were between 5 and 10; 8.3 per cent were between 10 and 15; 2.4 per cent were between 15 and 20, and 3.4 per cent were over 20.

Scarlet fever occurs chiefly in childhood, and, according to statistics, the largest percentage occurs in children under 10 years of age. The anatomy of the eustachian tube in the young favors the development of acute otitis media and its continuance into a chronic form. At birth, the eustachian tube measures not more than 20 mm. and is practically

5. Harries, E. H. R., and Gilhespy, F. B.: Scarlet Fever Otitis, Lancet **1**:843 (April 26) 1924.

straight. In the adult it measures about 36 mm., and its course is moderately tortuous. In the young, the pharyngeal orifice and caliber of the tube are quite as large as in the adult, and the pharyngeal orifice is nearer the floor of the nose than in the adult. These factors are supplemented by the effect of the faucial and pharyngeal tonsils, which will be discussed in a later paragraph.

Another factor that greatly predisposes to acute, and more particularly to chronic, suppurative otitis media is the concurrence of paranasal sinusitis. Contrary to the opinion of Layton,[6] I believe that sinusitis in scarlet fever is quite common.

There is a marked seasonal variation in the occurrence of scarlet fever and, therefore, of scarlatinal otitis. The season of the year in which the incidence is greatest usually begins about the middle of January and lasts until about the middle of May. There is, furthermore, a marked yearly variation in the number of patients with scarlet fever and scarlatinal otitis at the Philadelphia Hospital for Contagious Diseases. But the yearly variation in otitis media is more marked than that in the total number of patients. This variation among years is attributable almost wholly, it seems, to the difference in the virulence and severity of the different epidemics. Other factors, such as the age of the patient, the social status of the patient and the presence of tonsils and adenoids, remain fairly constant from year to year. Other variable factors, however, are the extent and virulence of concurrent epidemics of other exanthems, especially measles. It is my opinion, formed from experience and observation and not from definite statistics, that the concurrence of another exanthem with scarlet fever increases the incidence of aural complications, and that the increase is more marked in the case of measles than in the other exanthems.

It will be noted that the statistics offered here make no mention of acute catarrhal otitis media. It is more difficult to collect data on catarrhal than on suppurative conditions of the ear. It is not unlikely, however, that there is a catarrhal involvement of the middle ear in 50 per cent of patients with scarlet fever. In 1914, Borden[7] stated that in a series of autopsies on patients with scarlet fever, 94 per cent showed aural complications.

Of equal interest, and perhaps of greater practical interest, is the incidence according to the time in the period of morbidity of the onset of the otitic complication. Of the 2,186 ears affected with otitis media, in 415 the onset was in the first week of morbidity from scarlet fever; in

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6. Layton, T. B.: A Discussion on Otological and Rhinological Problems in Scarlet Fever and Measles, J. Laryng. & Otol. **39**:727, 1924.

7. Borden, C. R. C.: Aural Complications of the Exanthemata, Ann. Otol., Rhin. & Laryng. **23**:906, 1914.

533 it was in the second week; in 315, in the third week; in 245, in the fourth week; in 196, in the fifth week, and in 482, in the sixth week or later (table 4). These figures correspond fairly well with Gardiner's findings [4] in a series of patients in which the onset of otitis media in 82 occurred in the first week, in 64 in the second week, in 41 in the third week and in 27 in the fourth week.

The foregoing data support the assertion by Borden [7] that "it may truly be said of scarlet fever that aural complications of any degree of severity may arise at any time from the first day of the acute symptoms to the last day of convalescence;" with this opinion, I am in complete accord.

The average number of days of the discharge from ears that became dry while the patients were in the hospital was thirty-six, and the number of ears from which this average is derived is 1,915. This leaves a discrepancy of 271 ears, which may be accounted for by 196 patients

TABLE 4.—*Incidence According to Time in Period of Morbidity*

Week of Onset	Ears
1	415
2	533
3	315
4	245
5	196
6	482
Number of ears	2,186
Number of patients	1,535

with a discharge from the ear who were discharged from the hospital or who died. Of these patients, 121 had unilateral and 75 bilateral conditions. Forty-six of them underwent mastoidectomy, 37 unilaterally and 9 bilaterally.

Gardiner [4] reported that at the Edinburgh City Hospital, where no patient with otorrhea in scarlet fever was allowed to leave the hospital, the average stay in the hospital was sixty-eight days in 1919, sixty-two in 1920 and fifty-two in 1921.

The rôle of the faucial and pharyngeal tonsils in the etiology of aural complications in scarlet fever is widely recognized as an important one. Of the 1,535 patients with otitis media under discussion, only in 55, or 3.5 per cent, had these structures been removed prior to the development of scarlet fever. Of the total number of 14,733 patients with scarlet fever, the pharyngeal and faucial tonsils were absent in 861, or 6.8 per cent. The percentage is low in both groups, but it is noteworthy that it is about twice as high in those in whom aural complications did not develop as in those in whom they did develop. The high incidence of the presence of faucial and pharyngeal tonsils in those

patients in whom otitis media developed is better appreciated when cognizance is taken of the fact that 91.9 per cent of the patients were less than 10 years of age. Nevertheless, one who is familiar with the frequency with which tonsillectomy and adenoidectomy are performed among the children of this city who are admitted to the hospital wards through the free dispensaries would expect to find a higher incidence of tonsillectomy and adenoidectomy than is here found.

In this series of patients, there were 7 with meningitis, all streptococcic in type; 1 with abscess of the brain; 1 with lateral sinus thrombosis, and 1 with lateral sinus thrombosis and abscess of the brain. In all but 1 patient with meningitis the faucial and pharyngeal tonsils were present. All of the patients had mastoiditis requiring surgical treatment except 2 with meningitis, in whom, if it was present, it was not diagnosed. The mortality rate was 100 per cent. I am in accord with the opinion expressed by Borden [7] and Layton [6] to the effect that there is no great tendency of the disease to extend from the bone to the meninges.

The symptoms usually differ according to whether the complication comes on early or late. When the complication arises during the initial febrile period, it may be accompanied by a further elevation in temperature. Usually there are pain and restlessness, which more often come on during the late afternoon or evening, as pointed out by Layton.[8]

When the complication occurs late during the afebrile period, there may be a rise in temperature associated with pain and restlessness, but it often comes on so gradually, and without symptoms, that the first sign is a discharge on the pillow.

Occasionally, the infection passes through the middle ear, and the first sign is postauricular edema, redness or a subperiosteal abscess. The tympanic membrane in these ears shows a slight bulge, creamy white in appearance, with dilated vessels running from the center to the periphery. This is by no means uncommon.

The appearance of the tympanic membrane usually varies according to whether or not the otitis occurs during the febrile or the afebrile period. During the febrile period the tympanic membrane varies from a pink to a red bulging membrane, with all the landmarks obliterated, and when it is incised, a serous fluid is released.

The cases of otitis occurring later occasionally show desquamation in the external auditory canal, and the tympanic membrane is thickened, bulging and bluish white, and shows less evidence of acute inflammation; on incision, a thick, creamy pus is found.

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8. Layton, cited by Souper, H. R.: Scarlet Fever Otitis Media: Critical Review, J. Laryng. & Otol. **41**:233, 1926.

In some instances, when a discharge from the ear has reached a subacute stage, the tympanic membrane becomes edematous, loses its landmarks and presents, most often, it seems, in the posterior half, a nipple-like formation at the tip of which is a small perforation. This finding is not uncommon and signifies, as pointed out by Borden,[7] involvement of the mucus-lined spaces of the mastoid process.

I have not observed the early, rapid and extensive destruction of the tympanic membrane in scarlet fever which has been described in some textbooks on otology.

In discussing the diagnosis of mastoiditis in scarlet fever, the patients may be divided into two groups: those in whom it occurs early in the disease, and those in whom it occurs late.

Those patients in whom it develops early are likely to show fever, localized pain and tenderness, some sagging of the posterosuperior canal wall and a moderate or excessive discharge.

Those in whom it develops late are likely to present marked edema of the tympanic membrane, sometimes with a nipple-like perforation, sagging of the posterosuperior canal wall, a profuse and persistent discharge, usually no pain or fever and, frequently, moderate postauricular edema and redness, progressing rapidly to a subperiosteal abscess.

Discussion of roentgenographic and laboratory diagnosis is purposely omitted.

The useful principles of treatment which are available today are essentially the same as those available twenty years ago. They are embodied in the following outline:

(*a*) Prevention by keeping the nose and nasopharynx free from excessive secretions.

(*b*) Daily inspection of the ears by an otologist, with early myringotomy in every case in which the drum head is found bulging before it ruptures.

(*c*) Frequent cleansing of the external auditory canal to facilitate drainage.

(*d*) A consideration of the removal of the adenoids when the ear has discharged for three weeks.

(*e*) The performance of simple mastoidectomy in those cases in which there are persistent and profuse otorrhea, sagging of the posterosuperior canal wall, mastoid tenderness and edema with fever and positive roentgen evidence of mastoiditis requiring surgical treatment.

CONCLUSIONS

1. The incidence of otitis media in a series of 14,733 patients with scarlet fever was 10.8 per cent. In about one third of them the disease was bilateral. The incidence of mastoiditis requiring surgical treatment was 1.1 per cent.

2. The mortality in a series of 1,535 patients with scarlet fever with acute suppurative otitis media was 4 per cent. The mortality in a series

of 167 patients with scarlet fever on whom mastoidectomy was performed was 10.8 per cent.

3. Early incision of a bulging tympanic membrane and incision of a ruptured tympanic membrane when the rupture is inadequate for proper drainage tend to lower the incidence of acute mastoiditis requiring surgical intervention in scarlatinal otitis.

4. Repeated incision of a tympanic membrane is seldom of value.

5. Ninety-one and five-tenths per cent of the 1,535 patients with acute suppurative scarlatinal otitis media were children under 10 years of age.

6. The peculiarity of the eustachian tube in the child and the concurrence of pharyngeal and faucial tonsils and paranasal sinusitis predispose to the development of acute and chronic aural complications.

7. In scarlet fever, aural complications of any degree of severity may arise at any time, from the first day of the acute symptoms to the last day of convalescence.

8. The incidence of tonsillectomy and adenoidectomy in a series of 14,733 patients with scarlet fever was 6.8 per cent.

9. In otitis media in scarlet fever there is no great tendency for the disease to extend from the bone to the meninges, as shown by a total of 7 patients with meningitis from a series of 14,733 patients with scarlet fever.

10. Not infrequently the infection passes through the middle ear and appears as a postauricular edema, redness or a subperiosteal abscess, leaving the tympanic membrane intact.

11. The useful principles of treatment available today are essentially those that were available twenty years ago.

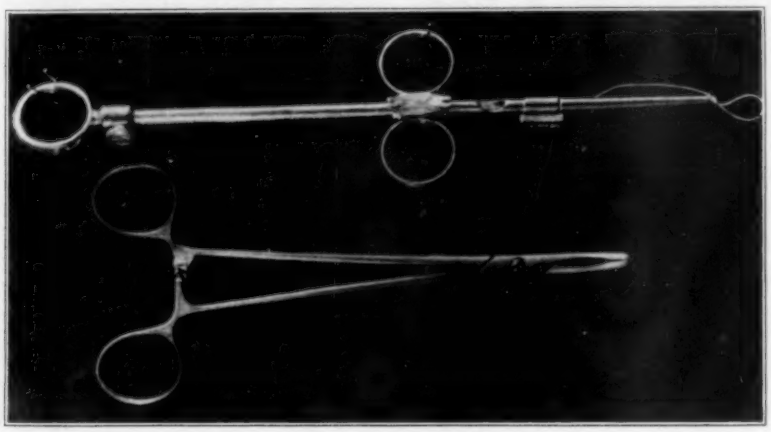
Clinical Notes

A NEW LIGATOR

NORMAN W. FILE, M.D., LYNCHBURG, VA.

The ligator described here is especially designed for tying bleeding points which are difficult to tie with the surgeon's fingers.

This instrument is composed of a main shaft into which a lower segment is welded. The segment is brought to a sloping point, in the end of which is a small opening slightly larger than a no. 2 catgut, through which the ligature is threaded. About 3 inches (7.6 cm.) from this tip there are another opening and a guide where the ligature makes its exit. On the under side of this portion of the shaft is a



A new instrument.

serrated flexible steel clip for attaching the ligator to the shank of a hemostat and receiving the loose ends of the ligature.

On the main shaft is a carriage, on which a clip is fastened to receive the ligature after it is brought through the exit. This also has two finger rests attached to it. The carriage works in a groove. At the lower end of the groove a small spring is fastened to hold the carriage in place while the ligature is being fastened around the clip. At the end of the main shaft a thumb rest is fastened, which can be removed for cleaning the instrument.

This instrument is used in the following way: A piece of no. 2 catgut about 18 inches (45.7 cm.) long is folded on itself, one end being slightly shorter than the other. The shorter end is carried around the loop three times and slipped through the circle formed by the wound ligature, thus making a triple loop slip knot, which is tied tightly, leaving a loop large enough above the knot to allow the point of the hemostat to pass through. The long end of the ligature is now threaded into the ligator as described and fastened to the clip on the carriage in the form of the figure eight. The hemostat is carried through the loop of the ligature in the end of the ligator. The ligator is then attached to one shank of the hemostat by

the flexible clip on the under side of the ligator, thereby enabling the operator to handle the ligator and hemostat with one hand, leaving the other hand free. The bleeding point is now picked up by the hemostat. The ligator is then detached from the hemostat and the loop carried around this point and drawn home in the same manner as in the manipulation of an Eave's snare, slight traction being used on the hemostat at the same time. Then the ligature is cut with scissors and the hemostat removed. In this way the bleeding vessel can be tied quickly without the surgeon's finger coming in contact in any way with the raw area.

The knot should be tied previously so that the ligatures can be put in alcohol a short time before being used. They can be quickly threaded into the ligator by the assistant.

I have used this instrument for two years in throat work with excellent results.

PARALYSIS OF A VOCAL CORD: PERIPHERAL TOXIC NEURITIS

CARROLL SMITH, M.D., SPOKANE, WASH.

The article by Drs. Gordon B. New and John H. Childrey on paralysis of the vocal cords, recently published in the ARCHIVES,[1] recalls two cases I have had within the past few months that I consider of sufficient interest to be reported. According to the etiologic classification in the article mentioned, these cases belong in the infectious group or are of the peripheral toxic neuritis type.

REPORT OF CASES

CASE 1.—On Dec. 1, 1931, Mrs. W., aged 44, the wife of a physician, came to me, complaining of attacks of choking when taking cold liquids, wheezing on exertion, some hoarseness during the past month and breaking of the voice when singing. Her health had always been good, and there was no previous illness to which the present condition could be attributed. However, she had recently had a "cold."

Examination showed an immobile right cord in the "cadaveric position" and some edematous swelling of the right ventricular band. The right antrum was somewhat dark on transillumination. The roentgenogram showed a cloudy right antrum; the other sinuses were normal. Examinations of the chest and roentgenograms of the chest and neck gave negative results. Examinations of the blood gave negative results, and the result of the basal metabolic test was minus 12. There was no enlargement of the thyroid gland. The antrum cleared up within two weeks under treatment with irrigations. Motion of the right cord was noticed within about six weeks, and recovery was complete within five months.

CASE 2.—On July 7, 1932, Mary W., aged 18, daughter of the patient in the preceding case, came in complaining of hoarseness for the past two months. Three

1. New, G. B., and Childrey, J. H.: Paralysis of the Vocal Cords: A Study of Two Hundred and Seventeen Medical Cases, Arch. Otolaryng. **16**:143 (Aug.) 1932.

months previously she was ill for a week with a septic sore throat (her tonsils had been removed); the temperature was high for several days, the maximum temperature being 105 F.

Examination showed the right cord in the "cadaveric position," with just perceptible movement. General examination, including all of the tests which had been made on the mother, gave negative results. The condition lasted two months, improvement being noticeable after six weeks, and resulted in complete recovery.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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PERORAL ENDOSCOPY

LOUIS H. CLERF, M.D.
PHILADELPHIA

Bronchoscopists are familiar with movements of the trachea and bronchi resulting from transmitted pulsatory motion and changes occurring during the respiratory cycle and cough. These observations have been made largely by direct inspection. A knowledge of these movements has contributed to an understanding of certain phenomena occurring in bronchial obstruction, namely, obstructive emphysema and obstructive atelectasis. In addition; the bronchoscopist's familiarity with bronchial movements aids him in the treatment in foreign body cases. There are, however, certain considerations with regard to movements of the bronchial tree that are not fully appreciated. The influence of bronchial movements, as described by Macklin,[1] on pathologic conditions and the effect of these on the bronchi are of interest to bronchoscopists. It is important to know that the lower end of the trachea is not fixed in position as some textbooks on anatomy state, but that it is loosely held and the bifurcation is freely movable. Normally the main bronchi and their associated structures are but loosely bound together and a certain range of movement is permitted which, although slight in any one part, totals a significant figure in the aggregate. From the standpoint of internal adjustments of the pulmonic mass and bronchi during respiration, the root zone is the important region, and movement here is marked because of the inspiratory elongation of the root. Macklin discussed changes in the bronchial tree and its relationship to the occurrence of tuberculosis. His views could be equally well applied to certain nontuberculous pulmonary diseases. Impairment of movement of the root of the lung could well result from an increase in the amount and density of connective tissue of the root and its extensions, particularly if accompanied by adhesions to the thoracic wall. Such a condi-

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From the Department of Bronchoscopy and Esophagoscopy, Jefferson Medical College.

1. Macklin, C. C.: The Dynamic Bronchial Tree, Am. Rev. Tuberc. **25**:393 (March) 1932.

tion might diminish or eliminate the normal elongation and retraction of the bronchial stems and might even influence the movement of the bronchial tree itself. Inflammatory conditions of the bronchial wall and peribronchial tissue with fibrosis would diminish the elasticity and expansibility of the tree. Peritracheal and peritracheobronchial lymphadenitis and lymphangitis might well cause restriction of movement. Macklin very properly propounded certain queries regarding the effects of disturbance in the movement of the bronchi. What influence would this have on getting rid of secretion? What would the effect be on the bronchial and pulmonary circulation and on lymphatic drainage? Would there be changes in the muscle and elastic tissue? He expressed the hope that his contribution would stimulate a study of these problems.

In the routine study of patients, certain clinical observations are often made which cannot be entirely explained. In his paper on obstructive pulmonary emphysema, Van Allen [2] pointed out that the emphysema is always lobar, involving one or more lobes, and is always associated with partial obstruction of the corresponding bronchus. The cause of failure of lobular bronchial obstruction to produce emphysema is assigned to collateral respiration. This function is ascribed to collateral connections that exist between the airways of the lobules throughout the lobe, and, in their studies, it was found that the lobular units of the bronchial tree were not independent of each other at the periphery; instead, they are joined together by minute openings between the alveoli where the interlobular septums are incomplete. These connections are utilized to convey air during respiration to and from a division of a lobe where the bronchus of that part is obstructed. Van Allen carried out a number of experiments which he has described. These showed that the condition could be reproduced in one or more lobes of the lung but not solely in lobular divisions. Applied clinically, it becomes clear why obstructive emphysema has been seen only with lobar distribution.

NEOPLASMS OF THE TRACHEA AND BRONCHI

Benign Neoplasms.—Papillomas in the air passages are most commonly found involving the larynx; occasionally they may be found on the soft palate, faucial pillars, tonsils or contiguous structures. Involvement of the trachea or bronchial tree is not commonly observed. There is also much speculation regarding the route of metastasis or reinfection in cases when the papillomas recur in new sites. In the case of papilloma reported by Hitz and Oesterlin,[3] metastasis of the growth to the

2. Van Allen, C. M.: Obstructive Pulmonary Emphysema and Collateral Respiration, Surg., Gynec. & Obst. **55**:303 (Sept.) 1932.

3. Hitz, H. B., and Oesterlin, E.: Case of Multiple Papillomata of the Larynx with Aerial Metastases to Lungs, Am. J. Path. **8**:333 (May) 1932.

bronchi apparently occurred by the aerial route. The patient, a child, aged 2 years, had difficulty with breathing and was aphonic. A diagnosis of papilloma of the larynx was made, and the growths were removed by direct laryngoscopy. There was a recurrence of the papilloma. Radium was applied on several occasions with apparently no outstanding beneficial results, for later, owing to the rapid recurrence of the growths, it became necessary to perform a tracheotomy. Death resulted following an unsuccessful attempt to change the tracheotomy cannula.

The postmortem findings were remarkable in that not only was the larynx and upper portion of the trachea entirely filled with the papillomatous masses, but there were found in the lungs many small cavities varying in size, some as large as hazelnuts. Many of these communicated with smaller bronchi. The walls of these cavities were covered with minute granules. The small areas in the lungs were at first thought to be tuberculous, but the histologic appearances were those of a neoplastic growth, and in many instances they were made up of stratified squamous epithelial cells exactly the same as were found in the papilloma of the larynx. Metastatic dissemination through lymph channels was ruled out by the study of the peritracheal and peribronchial nodes. Metaplasia as the possible explanation of the occurrence of these tumor masses did not seem to be a reasonable one. The writers believed that all of the tumors in the lung had a common origin, namely, that the tumor masses were detached from the larynx and carried into the bronchi with the inspired air. In this way they became implantation metastases and began to grow in the alveoli. In some areas there appeared to be a connective tissue reaction against the foreign material. If this is true, closer attention should be paid to the occurrence of this type of metastasis with the probability that more cases of this type will be observed.

In reviewing the literature of papilloma of the bronchus, Orton [4] found two authentic reports, although there were a number of cases of polyps reported. He added two additional cases to the literature and emphasized the importance of diagnostic bronchoscopy as an aid in obscure conditions of the chest. The patients, both children, had papilloma of the larynx, for which frequent laryngoscopic removals were performed with satisfactory results. Dyspnea later developed in both children. No papillomas were found by direct inspection of the larynx. Bronchoscopy was proceeded with, and in one case a mass of papilloma was found in the trachea at the bifurcation; in the other, a large papilloma was removed from the orifice of the left main bronchus. Orton

4. Orton, H. B.: Papilloma of the Bronchus, Ann. Otol., Rhin. & Laryng. **41**:933 (Sept.) 1932.

expressed the opinion that in both of these cases the papillomas were transplants from the original growth in the larynx and not primary in origin.

A growth that causes death is not, strictly speaking, benign, nor is a tumor-like inflammatory hyperplasia histologically a neoplasm. In the opinion of the Jacksons,[5] the clinician's primary concern in tumor-like nonmalignant obstructions of the bronchi is secondary atelectasis, bronchiectasis and pulmonary abscess caused by obstruction to ventilation and drainage and how to get rid of the obstruction. Benign growths of the tracheobronchial tree encountered bronchoscopically are as follows: angioma, hematoma, adenoma, myoma, myxoma, papilloma, fibroma, fibrolipoma, adenomatous polyp, lymphoma, lymphangioma, lymphadenoma, lipoma, ecchondroma, osteoma, chondrosteoma, tracheopathia osteoplastica, teratoma, retention cyst, amyloid tumor, aberrant thyroid, specific granuloma ([*a*] tuberculous, [*b*] syphilitic and [*c*] mycotic), nonspecific granuloma, granulation tissue and inflammatory hyperplasia. Many of these are of inflammatory origin and would not be classed histologically as true neoplasms. The most common of all growths encountered bronchoscopically is a tumor-like formation of inflammatory hyperplasia. Histologically, the lesion is not simply a chronic mucosal bronchitis, though, of course, the latter condition may coexist in the vicinity. The borderline between benign true neoplasms and inflammatory hyperplasia is often indistinct histologically. Clinically, the seriousness of the lesion depends more on the degree and the region of the obstruction it causes than on the histology of the growth. A papilloma at the bifurcation may kill the patient by asphyxia, and a tumor-like inflammatory hyperplasia in a bronchus may produce, in the tributary portion of the lung, atelectasis, drowned lung, pulmonary abscess, bronchiectasis and empyema, or all of these in succession. A recovery from these secondary conditions follows bronchoscopic removal of the growth, if the diagnosis is not made too late. The only way an early diagnosis of endobronchial growth can be made positively is by bronchoscopy. The only way to determine on the living patient that an endobronchial growth is benign is by histologic examination of a bronchoscopically removed specimen. Bronchoscopic removal of an endobronchial growth is a simple and safe procedure in trained hands. Removal of the obstructive growth reestablishes ventilation and drainage, and thereby the defensive power of the lung is restored. Reexamination within a few weeks or months is indicated after removal of a benign growth from a bronchus just as in the case of removal of similar growths elsewhere in the body.

5. Jackson, C., and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi, with Especial Reference to Tumor-Like Formations of Inflammatory Origin, J. A. M. A. **90**:1747 (Nov. 19) 1932.

Observations on benign tumors of the bronchus by Wessler and Rabin [6] were limited to the form known as adenomatous polyp, the most common type of bronchial tumor. In their opinion, the definitive diagnosis of benign tumor of the bronchus rests with bronchoscopic examination. Everything that has gone before may be suggestive or strongly presumptive, but only the bronchoscopic demonstration of the tumor furnishes a certain diagnosis. The tumor commonly appears globular or oval and may be sessile or attached by a pedicle, thus being freely movable. In their series of seventeen cases, ten were found in the bronchi of the lower lobe. The severity of the pathologic changes in the lungs bears a close relationship to the duration and degree of the bronchial occlusion. The treatment of benign tumor of the bronchus consists of removal of the tumor through the bronchoscope.

Malignant Neoplasms.—Malignant tumors involving the trachea are uncommon, and may be considered rare when compared with the occurrence of bronchial carcinoma. In the report of a case of primary carcinoma involving the lower third of the trachea, Gilfoy [7] discussed the diagnosis and the importance of direct examination. The symptom complex of intermittent dyspnea, cough and perhaps occasional hemoptysis in an apparently healthy patient, and the absence of obvious intrathoracic signs should suggest a tumor of the tracheobronchial tree. In such a case, direct examination is clearly indicated. Electrofulguration of the base of the growth in Gilfoy's case was supplemented by high voltage roentgen therapy. The patient was last observed sixteen months after a positive diagnosis had been made. He was then in good condition and had gained 25 pounds (11.3 Kg.) in weight.

In discussing the diagnosis of malignant pulmonary neoplasms, Edwards [8] emphasized the importance of bronchoscopy, particularly in cases of obstruction to a bronchus. The diagnosis can be made with certainty by removal of a portion of the growth.

Vinson [9] agreed that there is no better method of arriving at a conclusive diagnosis of primary carcinoma of the bronchus than bronchoscopic removal of a specimen of tissue. He emphasized the difficulties encountered in arriving at a diagnosis by considering the history, gen-

6. Wessler, H., and Rabin, C. B.: Benign Tumors of the Bronchus, Am. J. M. Sc. **183**:164 (Feb.) 1932.

7. Gilfoy, F. E.: Primary Malignant Tumors of the Lower Third of the Trachea, Arch. Otolaryng. **16**:182 (Aug.) 1932.

8. Edwards, A. T.: The Surgical Treatment of New Growth, Brit. M. J. **1**:827 (May 7) 1932.

9. Vinson, P. P.: The Necessity of Bronchoscopic Examination in Distinguishing Primary Carcinoma of the Bronchus from Suppurative Diseases of the Lungs, Surg., Gynec. & Obst. **54**:105 (Jan.) 1932.

eral examination or even roentgen examination. A number of cases are cited for the purpose of illustrating some of the diagnostic difficulties.

Davidson [10] emphasized the fact that diagnosis of intrathoracic new growths has become a complicated affair, and that often history and physical and roentgen examinations are inadequate. He stressed the importance of team work, and the need of an appropriate group consisting of physician, roentgenologist and bronchoscopist. In discussing the bronchoscopic diagnosis, he believed that the value of bronchoscopy was chiefly seen in those cases in which definite roentgen evidence is lacking. This is best illustrated in cases of hemoptysis. Additional aid consists in the removal of tissue from the growth. This is of value only in endobronchial neoplasms. In regard to treatment, he expressed the opinion that one must look to the surgeon and the bronchoscopist for the actual technic of any radical therapy that may be attempted. Much information may be secured by bronchoscopy relative to the accessibility of growths for surgical extirpation or for endobronchial treatment.

With an increase in the number of cases of pulmonary carcinoma that are being recognized in the living, Manges [11] stressed the need of considering cancer as a diagnostic possibility in all obscure conditions of the chest and emphasized the importance of becoming "primary-carcinoma-of-the-lung-minded." Obviously one should have a roentgen study of the chest made if there is a question in diagnosis. Bronchoscopy can supplement the diagnosis, and in cases of carcinoma of the larger bronchi tissue may be removed for histologic study. In those cases in which the growth does not extend into the larger bronchi, the bronchoscopist often can make a tentative diagnosis of malignancy by observing fixity, rigidity and deformity of the bronchial wall. This information, taken in conjunction with the data secured from the history, physical examination and roentgen study, may justify a positive diagnosis. In a series of thirty-six cases selected for roentgenographic description, twenty-nine exhibited bronchoscopic evidences of new growth. Manges expressed the belief that bronchoscopy with biopsy has contributed more to the knowledge of pulmonary carcinoma than any other one procedure. This should never be omitted because of the positive data secured and the absence of any untoward effects. A report is made regarding the methods of treatment and the results secured to date.

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10. Davidson, M.: Intrathoracic New Growths and the Value of Bronchoscopy in Diagnosis and Treatment, Brit. M. J. **2**:617 (Oct. 1) 1932.

11. Manges, W. F.: Primary Carcinoma of the Lung: Roentgen Diagnosis and Preliminary Report in Roentgen Therapy, Am. J. Roentgenol. **27**:858 (June) 1932.

In considering the importance of bronchoscopy as a diagnostic aid, a significant fact in this connection is that by bronchoscopic investigation of the tracheobronchial tree a positive diagnosis of cancer has been made during life in a large number of cases. In certain of these it has been possible to remove the growth and destroy its point of attachment to the bronchial wall with gratifying results. Whether all of these cases are truly malignant remains a question. Further study from a clinical and pathologic standpoint will probably settle this matter. In 1932, I [12] again pointed out what is probably more clearly appreciated by the bronchoscopist than the internist, namely, the influence of bronchial obstruction in these cases. Often the symptoms indicate the presence of marked pulmonary suppuration, which in many instances has been the cause of a fatal termination. Although bronchial neoplasms are commonly observed in the larger bronchi and are accessible to bronchoscopic investigation, one occasionally observes narrowing of a bronchus with fixity, rigidity and distortion. In the presence of corroborative data secured by general examination and roentgen study, a positive diagnosis of cancer can be made and appropriate treatment can be proceeded with without waiting for metastasis or other corroborative data.

In the opinion of Vinson and Leedy,[13] direct treatment of carcinoma of the bronchus by fulguration or diathermy through a bronchoscope seems ill advised in the majority of cases, since the growth usually involves one of the main bronchi and extends well beyond the confines of the wall of the bronchus. Commonly the appearances on bronchoscopic examination are not an accurate indication as to the size of the growth. Additional methods of treatment are the employment of radium or high voltage roentgen therapy. In a report of seventy-one cases of primary malignant disease of the tracheobronchial tree in which the diagnosis was made by bronchoscopy with biopsy, it was found that sixty-one patients are dead. Of these, thirty-two received roentgen treatment. Those receiving treatment lived slightly more than eight months. The untreated patients, a total of thirty-one, lived on an average of little more than five months. Ten patients who were treated by high voltage roentgen therapy are improved and are living and apparently well, the average duration being twenty-three months. Abstracts of the cases of the ten living patients are presented.

Metastasis of a hypernephroma into the lumen of a bronchus and the occurrence of signs and symptoms simulating a bronchial neoplasm

12. Clerf, L. H.: Bronchoscopic Aids in the Diagnosis of Pulmonary Disease, M. Arts & Indianapolis M. J. **35**:423 (July) 1932.

13. Vinson, P. P., and Leedy, E. T.: Roentgen Treatment of Primary Malignant Diseases of the Tracheobronchial Tree, Ann. Otol., Rhin. & Laryng. **41**:1259 (Dec.) 1932.

are of rare occurrence. Vinson and Martin [14] reported such a case occurring in a man, aged 58, who had intermittent hematuria of six years' duration with an absence of respiratory symptoms. A diagnosis of hypernephroma was made and operation performed. About two years later, he returned complaining of respiratory symptoms, notably cough, occasional hemoptysis and indefinite pain in right axilla. At bronchoscopy, an ulcerated bleeding tumor was observed occluding the bronchus of the right lower lobe. Tissue removed was found to be adenocarcinoma of a hypernephroma type.

BRONCHOSCOPY IN DISEASE

The advantages of the employment of methods of direct examination which can be safely applied are obvious and are generally utilized in diagnosis. Bronchoscopic examination is an accepted procedure. The extent of its use as a diagnostic aid is in great part dependent on a knowledge of the indications for its employment.

Although the literature is replete with observations regarding the diagnosis and treatment of bronchial obstruction in adults and older children, Lierle [15] directed attention to a relative scarcity of data concerning bronchial obstruction in infants and younger children. He reported the occurrence of bronchial obstruction in two young children. The histories were inconclusive. A clinical diagnosis of bronchial obstruction was made on the basis of the physical signs and roentgen findings. In one case there was obstructive atelectasis; in the other, there was obstructive emphysema. At bronchoscopy there was found in each case a small inflammatory mass in the bronchus with suppurative changes distal to the obstruction. Lierle concluded that neoplasms and congenital anomalies causing obstruction in infants are rare; that pressure from tuberculous lymph nodes must always be kept in mind as diagnostic possibilities, and that infants and young children with stationary or progressive chronic pneumonia should be given the benefit of bronchoscopic study, particularly when there is evidence of atelectasis or emphysema.

In presenting the subject of direct examination of the air passages of the infant, Tucker [16] emphasized the importance of study of the infant larynx in respiratory and throat diseases and encouraged the otolaryngologist to acquire the technic for direct laryngoscopy. The

14. Vinson, P. P., and Martin, W. J.: Pulmonary Metastasis from Hypernephroma Diagnosed by Bronchoscopy, Arch. Otolaryng. **15**:368 (March) 1932.

15. Lierle, D. M.: Bronchial Obstruction in Infancy, Laryngoscope **42**:627 (Aug.) 1932.

16. Tucker, G.: The Infant Larynx: Direct Laryngoscopic Observations, J. A. M. A. **99**:1899 (Dec. 3) 1932.

most important measurement of the larynx is the subglottic diameter for the reason that this area is completely surrounded by cartilage, the cricoid ring. In the normal larynx this cannot be enlarged by instrumentation or by passage of a large bronchoscope without trauma. The introduction by sight of a special infant bronchoscope for the relief of urgent laryngeal dyspnea and the performance of tracheotomy and putting the diseased larynx at rest will conserve the laryngeal structures more effectively than intubation. He concluded that complete examination of the infant larynx can be made only by the direct method. The difficulties of the examination are more apparent than real if proper preparation, instruments and technic are used.

Accuracy in diagnosis must always be the primary aim of every physician. Without a correct diagnosis treatment cannot be scientifically applied. In regarding the results of their investigations in a series of 450 bronchoscopies carried out in their clinic during the past year, Pinchin and Morlock [17] agreed that bronchoscopic examination is a valuable additional method in the investigation of pulmonary disease; in neoplasm and suppurative conditions, it is essential. The method is safe in experienced hands and does not unduly disturb the patient. In their series of cases no ill effects were observed, nor have patients complained.

In spite of a progressive increase in the number of endoscopic procedures being performed generally, Peery [18] agreed that there is lacking a proper appreciation of the indications, limitations and accomplishments of these methods. His report of four hundred procedures which included sixty-seven foreign body cases afforded ample evidence of the work that can be carried out in a small hospital with the achievement of definite results and without mortality.

In the opinion of Negus,[19] the value of bronchoscopy in diagnosis and treatment cannot be overestimated. Because of a relatively small number of foreign bodies met with in the average clinic, one's experiences would be small; the bronchoscopist must therefore be willing to devote a considerable amount of his time to the whole range of endoscopic methods, particularly in the examination and treatment of patients suffering from diseases of the chest other than foreign body obstruction. In the treatment of abscess of the lung it is obviously of the greatest importance that bronchoscopy should be employed in all cases, if only for the exclusion of the presence of a foreign body. Results

17. Pinchin, A. J. S., and Morlock, H. V.: The Bronchoscope in the Diagnosis of Pulmonary Disease, Lancet **1**:224 (Jan. 30) 1932.

18. Peery, V. P.: Report of Four Hundred Endoscopic Procedures Without Mortality, South. Med. & Surg. **94**:516 (Aug.) 1932.

19. Negus, V. E.: Value of Endoscopic Methods in Upper Air Passages, Lancet **2**:169 (July 23) 1932.

obtained by bronchoscopy are so satisfactory that this method should be tried preferably in the early stages of the disease.

In a general discussion of the value and advantages of methods of direct examination in the diagnosis and treatment of diseases of the larynx, trachea, bronchus, lungs and esophagus, Tucker [20] stated that bronchoscopy (peroral endoscopy) is now so perfected in technic that its most important use is in the diagnosis and treatment of disease of the air and food passages. Foreign body work, for which it was originally designed and in which it still will give a cure in 98 per cent of the cases, is only about 1 per cent of the work in bronchoscopy. Bronchoscopy is more closely related to otolaryngology than any other specialty. The technic can be acquired by any physician who will give the necessary time and who will properly organize a clinic. As a life-saving procedure in the diagnosis and treatment of disease in its special field, it is second to no other procedure and should be available to the medical profession in every large community.

The experiences of Hart and Davis [21] corroborated those of other observers, notably Moersch and Boothby, regarding the value of oxygen therapy in dyspnea developing after bronchoscopy. In their experience, the use of an oxygen tent following bronchoscopy will often avoid the necessity for tracheotomy in uncomplicated postbronchoscopic laryngeal edema.

Postoperative Pulmonary Atelectasis.—In a discussion of postoperative pulmonary complications, Butler [22] enumerated and briefly discussed the primary and secondary factors responsible for these and ventured the opinion that atelectasis is the result of a parasympathetic viscerovisceral reflex. He believed that the mere passage of a bronchoscope gave rise to mechanical stimulation which could modify or inhibit pulmonary reflexes. In discussing the rôle of early bronchoscopy in preventing, aborting or relieving some of these complications, it was emphasized that bronchoscopy could not abort or relieve postoperative pulmonary complications due primarily to embolism, cardiac failure or infection via direct channels. It may, however, if utilized early, be of great value in the prevention or treatment of complications due primarily to tracheal aspiration, pulmonary reflexes, hypoventilation or flooding of lungs by purulent secretions. In his experience, operative wounds of from one day to three weeks' standing have suffered no ill effects as a result of bronchoscopic treatment.

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20. Tucker, G.: Observations on Bronchoscopy (Peroral Endoscopy), Pennsylvania M. J. **35**:782 (Aug.) 1932.

21. Hart, V. K., and Davis, S. W.: Oxygen Tent in Postbronchoscopic Care of Children, Arch. Otolaryng. **16**:522 (Oct.) 1932.

22. Butler, E. F.: Bronchoscopic Relief of Postoperative Pulmonary Conditions, Ann. Otol., Rhin. & Laryng. **41**:1221 (Dec.) 1932.

Bronchial Stenosis.—In a very complete and comprehensive presentation on bronchial stenosis, Eloesser [23] discussed the value of bronchoscopy in diagnosis and treatment. In his opinion, the bronchoscope is invaluable in the diagnosis of obstruction of the lower and middle lobes. In the more central stenoses, it reveals the site, degree and sometimes the extent and nature of the constriction. It can demonstrate the presence of intrabronchial tumors, ulceration and scars, extrabronchial pressure and deviations of the trachea and larger bronchi. Information concerning the bronchi of the upper lobe is less readily secured because of inability to look around the corner with a rigid tube. The orifice of a bronchus of the upper lobe may be inspected and inflammatory changes and the presence of tumors detected if these extend to the main bronchus. The presence of pus or blood draining from an orifice of the upper lobe bronchus may also be detected. If, therefore, the stenosis lies beyond the orifice of the bronchus, it is not visible and all collateral information will permit of deductions. In the treatment of some forms of stenosis, bronchoscopy must be considered. In certain strictures, bronchoscopic dilatation at intervals affords great relief. Stenosis caused by aspirated foreign bodies or by endogenous material can best be treated by bronchoscopy. Benign growths may be removed bronchoscopically or destroyed by electrocoagulation through the bronchoscope.

Following a general discussion of pulmonary abscess following tonsillectomy, the general plans of treatment and the very satisfactory results secured by bronchoscopic therapy, Soulas [24] commented on the favorable effects of bronchoscopy. He directed attention to the fact that the bronchoscope is not introduced directly into the abscess as it cannot be passed beyond the secondary bronchi. The effect of bronchoscopy is far more complex, and the mechanism of its action is not wholly understood. Larger bronchi are easily reached directly; obstructing secretions are aspirated and bronchial permeability reestablished. In Soulas' opinion it is a mistake to assume that aspiration is the sole purpose of bronchoscopy in these cases. That is why he prefers the term bronchoscopic therapy to that of bronchoscopic aspiration, aspiration being only one of the effects obtained. Often but a few cubic centimeters of secretion is aspirated, but drainage continues for hours or even days afterward as shown by roentgen study and other findings. Thus drainage causes a reestablishment of pulmonary aeration. The effect and intimate mechanism of this action are not clearly understood.

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23. Eloesser, L.: Bronchial Stenosis, J. Thoracic Surg. **1**:270 (Feb.); 373 (April); 485 (June) 1932.

24. Soulas, A.: Pulmonary Abscess Following Tonsillectomy: The Rôle of Bronchoscopy, Rev. de laryng. **7**:72 (July-Aug.) 1932.

There is every reason to believe that the action is most important and finds its expression in a change of bacterial flora and general improvement in the patient's condition. Soulas considered bronchoscopy a relatively simple procedure which is nontraumatizing and is well borne at all ages.

In discussing the treatment of pulmonary abscess by bronchoscopy, Kearney [25] stated that patients for whom bronchoscopy would be lifesaving are deprived of its benefits by the unnecessary fear shared by many physicians as well as patients that bronchoscopy is an exceedingly dangerous procedure. He agreed that properly performed bronchoscopy is without mortality.

The study of Kramer and Glass [26] was based on a correlation of bronchoscopic, operative and autopsy findings and anatomic studies with a view of securing information regarding the relation of pulmonary abscess to the overlying thoracic cage. In surgical treatment of abscess, the importance of accurate localization is obvious. They emphasized the need of adopting an accepted nomenclature for the various accessible bronchial branches that are bronchoscopically important so that bronchoscopic reports would be generally understood. In their study the lobar bronchial divisions were first studied and their constancy noted; the segments were then delineated on the pulmonary surfaces by the injection of fluid dyes into these bronchial divisions. In eighty pairs of human lungs studied, the bronchial divisions in 85 per cent were recorded. In this way it was possible to divide each lobe in various segments corresponding to fairly constant bronchial divisions. A number of case records were cited to illustrate some of the problems arising in the diagnosis and treatment of pulmonary abscess with relation to their localization.

Bronchiectasis.—In a number of papers by Ballon and others [27] in which the subject of bronchiectasis was thoroughly presented the question of bronchoscopy in diagnosis as well as in treatment was considered. In diagnosis, bronchoscopy was considered an important aid for the following reasons: It enables one to rule out the presence of a foreign body, new growth or bronchostenosis. It permits one to observe which bronchi are discharging pus. It allows of the most complete injection of iodized oil, 40 per cent, since it is possible to aspirate secretions and remove granulations before introducing the oil. By this method one can also direct the iodized oil to the desired area. Most

25. Kearney, H. L.: Treatment of Lung Abscess by Bronchoscopic Drainage, South. M. J. **25**:1019 (Oct.) 1932.

26. Kramer, R., and Glass, A.: Bronchoscopic Localization of Lung Abscess, Ann. Otol., Rhin. & Laryng. **41**:1210 (Dec.) 1932.

27. Ballon, H.; Singer, J. J., and Graham, E. A.: Bronchiectasis, J. Thoracic Surg. **1**:297 (Feb.); 397 (April) 1932.

important of all, it permits of a more exact interpretation of the picture produced by injection. It also allows one to exclude many of the causes of such an unexplained symptom as hemoptysis. It is possible to obtain uncontaminated cultures by this method. In the treatment of bronchiectasis, bronchoscopy was considered under operative procedures. The value of bronschoscopy in the treatment of bronchiectasis of foreign body origin and in certain cases resulting from partial bronchial obstruction is obvious. The chief object in treatment in a majority of cases of bronchiectasis is to promote drainage. While postural drainage satisfactorily accomplishes this in many cases, it is doubtful whether postural drainage can as completely empty the dilated bronchus as bronchoscopic aspiration. This can be verified by injecting the bronchi of a patient suffering from bronchiectasis by passive methods after postural drainage and by the bronchoscopic method after bronchoscopic aspiration. It is important to remember that neither postural drainage nor bronchoscopy will actually cure many patients with bronchiectasis; they can, however, cause an arrest of symptoms in certain cases. The value, therefore, of bronchoscopy in the treatment of bronchiectasis lies in removing or treating local causes, in promoting drainage and in attempting to prevent progression of the disease.

In the experience of Pinchin and Morlock,[28] bronchoscopy supplementing medical treatment in acute cases of pulmonary suppuration seemed to yield a definitely higher percentage of cures than with medical treatment alone. In a group of seventeen patients with chronic pulmonary suppuration who did not respond to medical treatment, a cure was secured in nearly every instance by bronchoscopic drainage. There was no mortality. In the hands of Pinchin and Morlock these conservative measures have given better results than surgical treatment. Their surgical statistics gave a mortality rate of 35 per cent with 65 per cent cures. Of the patients who have been cured, some had been left with sinuses which required several plastic operations. In bronchiectasis and bronchiectatic abscesses, bronchoscopic drainage, though not curative, is of great value as a palliative.

In discussing bronchiectasis, Warner [29] expressed the views generally held by bronchoscopists that bronchoscopic examination is indicated in order to ascertain if there is bronchial obstruction. He, however, questioned the advisability of routine bronchoscopic examination, because he felt that bronchoscopy is not devoid of danger. He believed that postural drainage could well supplant bronchoscopic aspiration in those

28. Pinchin, A. J. S., and Morlock, H. V.: Bronchoscope in Treatment of Pulmonary Suppuration, Lancet **2**:604 (Sept. 17) 1932.

29. Warner, W. P.: Bronchiectasis: Aetiology, Diagnosis and Treatment, Canad. M. A. J. **27**:583 (Dec.) 1932.

cases in which no obstruction exists. It might, however, be necessary to resort to bronchoscopy when obstruction was present in order either to remove the obstruction or to aid in the aspiration of secretion.

Sergent [30] recommended that bronchiectasis be classified into three broad groups, as indicated in the title. He has found bronchoscopy useful for treatment in certain of these cases of bronchiectasis and abscess. Its object is to evacuate pus from those areas that drain poorly. A cure can be secured only in recent cases without pulmonary fibrosis. In chronic cases the results from bronchoscopy are palliative only, but this measure is definitely indicated if surgical treatment is considered. Preoperative bronchoscopy aids in draining pus, particularly in cases of putrid abscesses.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES

Pins at the periphery of the lung, referred to by the Jacksons,[31] constitute a class distinct from those lodged in the larger bronchi, which are relatively easy to remove bronchoscopically. Pins at the periphery of the lung are practically always lodged head downward. This may be due to the catching of the point and the tumbling over of the head end, and partly to the fact that the head end is the heavier. The primary cause of pins in the lung is carelessness in putting pins in the mouth. The numerous secondary etiologic factors are the same as those given in a previously published analysis. The cause of the pin reaching the periphery is a pawl and ratchet-like action of the pin; the head is free to move downward during inspiratory elongation of the bronchi, and the point catches and resists upward movement during the bronchial shortening of expiration and cough. The limit of downward travel is the smallest bronchus that the head of the pin can enter. After the initial choking, gagging and coughing, there is, in the case of pins at the periphery of the lung, a symptomless interval of a number of months. Sooner or later, however, suppurative changes with productive cough and progressively increasing impairment of health supervene, and a fatal ending may eventually occur if the pin is not removed. All pins at the periphery of the middle and lower lobes and descending branches of the upper lobes can be removed through the mouth by peroral costophrenic bronchoscopy. The ascending branches of the upper lobes present great difficulties, but fortunately their invasion by pins is exceedingly rare.

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30. Sergent, E.: Abscessed Bronchiectasis, Bronchiectatic Abscesses and Abscesses Causing Bronchiectasis, Presse méd. **40**:273, 1932.

31. Jackson, C., and Jackson, C. L.: Pins at the Periphery of the Lung, Arch. Otolaryng. **15**:860 (June) 1932.

In a summarized report of one hundred foreign body cases, Knauer's [32] statistics checked closely with those of other endoscopists. He found that the frequent use of atropine hypodermically following bronchoscopy has often averted tracheotomy. Atropine alone is used in children one hour before operation.

The occurrence of successive foreign bodies in the respiratory tract of the same patient is unusual in the experience of bronchoscopists. Peeler and Hart [33] reported such a case. The patient, a child, was admitted with the history of having choked on a coffee bean six weeks before. The case proved to be one of partial obstruction to the right bronchus by a whole coffee bean. This was removed bronchoscopically. The child made an uneventful recovery. Nine months later the child was again admitted with the history of having choked on a foreign body. Half of a white bean was removed from the right main bronchus. Prompt recovery followed its removal.

In the postoperative care of infants and young children, certain problems are encountered which are not often seen in the older child. Arbuckle [34] discussed the need of considering the findings and selecting the type of operation. In the presence of shock with cyanosis and dyspnea supportive treatment is imperative. A tracheotomy set should always be available for immediate use. The application of suction through the tracheotomy tube is a most satisfactory method of supplying lung drainage and of freeing the airway of obstruction. If one is unsuccessful by this method, the bronchoscope should be employed, even though it be necessary to do this repeatedly. The secretion of mucus should be encouraged rather than discouraged. This will be aided by the free administration of fluids either by mouth or by parenteral methods. No sedatives should be given. Expectorants are indicated. No drugs should be given which dry up the secretion. The oxygen tent is a most important adjunct, by the use of which many lives will be saved.

Perforation of a bronchus by pathologic lesions is not common. Perforation of a bronchus during instrumentation has no doubt occurred, although no cases are recorded. Myerson [35] reported a case in which the bronchus was perforated and the right pulmonary vein torn during an attempt to remove an impacted bronchial foreign body

32. Knauer, W. J.: Foreign Bodies in the Food and Air Passages: Report of One Hundred Cases, J. Florida M. A. **18**:126 (March) 1932.

33. Peeler, C. N., and Hart, V. K.: Successive Bronchial Foreign Bodies, South. M. J. **25**:729 (July) 1932.

34. Arbuckle, M. F.: Postoperative Care of Foreign Body in the Lung in Infants and Young Children, South. M. J. **25**:456 (May) 1932.

35. Myerson, M. C.: Perforation of the Bronchus with Notes on Bronchoscopic Anatomy, Ann. Otol., Rhin. & Laryng. **41**:1201 (Dec.) 1932.

of long sojourn. Packing directed to stop bleeding was introduced through the pulmonary vein into the left auricle, where it was found at autopsy. The patient was suffering from severe chronic cardiac disease which unquestionably influenced the proximity and size of the pulmonary vein. The bronchial walls were found to be the site of acute and chronic purulent inflammatory changes. Myerson made anatomic studies to determine the relationship of the trachea and bronchi to the heart and great vessels and recorded his findings.

In an analysis of the records of 243 foreign body cases in which treatment was given during a ten year period, Hart [36] and others found that in 15 per cent of cases with a definite history of a foreign body none was found, although in a considerable number there was evidence of a previous foreign body as indicated by traumatized mucosa in the esophagus or infection of the bronchial tree. A low mortality rate, 2.9 per cent, was reported, which included deaths from all causes.

Commenting on his review of a series of foreign bodies in the food and air passages, Cummings [37] agreed that about 70 per cent of foreign bodies found in adults occur in those wearing dentures. Flat objects of fair size commonly lodge in the esophagus at the level of the suprasternal notch.

In reporting a group of sixty-seven cases of foreign body impacted in the esophagus during a period of ten years, Diggle [38] remarked that he had observed only two verified cases of foreign body in the lower air passages. He observed that foreign bodies in the air passages were more frequent in America and attributed this to the habit of eating peanuts. Although no time should be lost in definitely establishing its existence if foreign body is suspected, he does not believe that the urgency in these cases is so great as to necessitate action before roentgen studies are carried out or the services of an endoscopist secured. He deprecated the use of blind methods of removal.

In a report of a series of foreign body cases, Miller [39] commented on certain phases of the problem of removal and emphasized the importance of remembering that esophagoscopy is much more difficult and dangerous to perform than bronchoscopy, especially in infants and in the aged. Persons wearing dentures have difficulty in detecting foreign

36. Hart, V. K.; Peeler, C. N., and Motley, F. E.: A Review of Ten Years' Experience with Foreign Bodies in the Throat, Lungs and Esophagus, South. Med. & Surg. **94**:137 (March) 1932.

37. Cummings, G. O.: Foreign Bodies in the Food and Air Passages, J. Maine M. A. **23**:44 (March) 1932.

38. Diggle, F. H.: Foreign Bodies in the Esophagus, Brit. M. J. **1**:277 (Feb. 13) 1932.

39. Miller, J.: Extraction of Foreign Bodies from the Air and Food Passages by Direct Methods, Arch. Otolaryng. **16**:188 (Aug.) 1932.

bodies, especially bones, and often swallow them. In perforation of the cervical esophagus, when one is sure of the perforation, he advised prophylactic mediastinotomy through the neck, Marshek's method, while in a perforation lower down in the esophagus, enlargement of the opening for mediastinal drainage and feeding the patient via a nasal tube, Seiffert's method, were advocated.

It was interesting to note that of twenty-four esophageal foreign bodies removed esophagoscopically by Alcantara and Ayuyao,[40] there were fifteen cases in which coins were found, two cases in which a common pin was located and one case in which the foreign object was a safety pin. In twenty-one cases the foreign body lodged at the cricopharyngeal constriction.

In the case reported by Tucker,[41] the foreign body, a safety pin, had been missed about a month before the patient came under his care. When admitted, the child's condition was fairly good. There was a history of vomiting a considerable quantity of blood. A roentgen study before admission revealed a safety pin in the esophagus; eight hours later it was found to be in the stomach. No attempt was made to remove the foreign body, nor was instrumentation carried out at any time. The foreign body made satisfactory progress through the alimentary canal and was passed spontaneously. During this time, however, the patient became progressively worse with increase in anemia and a fatal termination occurred after a profuse hemorrhage. Post mortem, a tear was found in the wall of the esophagus with a tear in the wall of the aorta contiguous to it, together with an acute purulent pericarditis and multiple embolic abscesses in the kidney. This case emphasized the importance of roentgen study of the esophagus in every patient who has disturbances with swallowing and the need of teaching prophylaxis in foreign body accidents.

In their presentation on perforations of the cervical esophagus, Heatley and Pearse [42] discussed the management of these cases, reviewed the anatomy, the symptoms and the diagnosis and briefly summarized the opinions of other observers concerning the treatment. The case histories of a series of eight patients were presented and comments were made concerning the care and progress. The technic of external operation was discussed.

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40. Alcantara, V. C., and Ayuyao, C. D.: Foreign Bodies in the Esophagus, J. Philippine Islands M. A. **12**:31 (Jan.) 1932.

41. Tucker, G.: Spontaneous Perforation of the Aorta from an Open Safety Pin in the Esophagus, with Fatal Hemorrhage, Occurring in a Child, Seven Months of Age, During the Passage of the Pin Through Esophagus and Gastro-Intestinal Tract, Ann. Otol., Rhin. & Laryng. **41**:1228 (Dec.) 1932.

42. Heatley, C. A., and Pearse, H. E.: The Management of Perforations of the Cervical Esophagus, Ann. Otol., Rhin. & Laryng. **41**:1233 (Dec.) 1932.

The studies of Stokes and Wilder [43] were undertaken to ascertain whether the loss of saliva in atresia of the esophagus is the most important factor either in the failure to gain weight and height or in the fatal outcome. Serious effects on nutrition resulting from closure of the esophagus might be explained on the basis of a salivary gland hormone necessary to life and growth or a loss to the body of certain mineral salts secreted in the saliva; finally, the lack of saliva or other secretions above the esophagus may in some way interfere with normal gastric and intestinal function. The writers carried out a series of observations on a patient with complete obstruction of the esophagus of seven years' duration and concluded that a well balanced diet is most important in maintaining nutrition and promoting growth. The addition of saliva and other secretions of the mouth to the food is probably not necessary for the maintenance of a normal state of nutrition. This conclusion was supported by experimental studies on pigs in which esophageal fistulas were created. Growth in these animals proceeded at a rapid rate.

Various methods of treatment of impermeable stricture of the esophagus were discussed by Iglauer,[44] who reported a case of atresia of the esophagus in which the barrier was penetrated by retrograde introduction of a urethral catheter passed through a cystoscope. The injection of opaque oil through the retrograde catheter combined with introduction of oil from above enabled him to secure a roentgen film showing the thickness of the stricture. Iglauer expressed the opinion that retrograde introduction of a urethral catheter should be of considerable value in the study and treatment of atresia of the esophagus.

A thorough search of the literature of benign esophageal neoplasms convinced Patterson [45] that these growths are rare. A total of sixty-one cases were recorded from 1717 to 1932. The most common growth found was polyp, with papilloma and myoma next in order of frequency. Benign growths are commonly single and are practically always pedunculated. Patterson [45] reported the case of a man, aged 54, who for six months had been troubled with a growth in the throat which at times would come up on his tongue. The growth was removed esophagoscopically. The pedicle was found to be attached to the left side of the esophagus about 2 cm. below its upper end; it was very tough and was

43. Stokes, J. J., and Wilder, T. S.: Esophageal Obstruction: Clinical and Experimental Study of Its Effects on Life and Growth, Am. J. Dis. Child. **43**:604 (March) 1932.

44. Iglauer, S.: Impermeable Stricture of the Esophagus Relieved by Retrograde Bouginage with the Aid of a Cystoscope Inserted Through a Gastrostomy, Ann. Otol., Rhin. & Laryng. **41**:1191 (Dec.) 1932.

45. Patterson, E. J.: Benign Neoplasms of the Esophagus: Report of a Case of Myxofibroma, Ann. Otol., Rhin. & Laryng. **41**:942 (Sept.) 1932.

severed with difficulty. The growth was 7 cm. in length. The histologic diagnosis was myxofibroma. Patterson concluded that benign neoplasms may occur in any part of the esophagus and are frequently asymptomatic, therefore unsuspected. They are practically always pedunculated, allowing of great mobility. Conclusive differential diagnosis between benign and malignant neoplasms can be made only by esophagoscopy.

In the experience of Beck and Guttman,[46] the absolute diagnosis of endo-esophageal carcinoma rests on esophagoscopy and biopsy. Esophagoscopic examination is performed, whether roentgen evidence of esophageal disease is present or not, to ascertain the nature of the pathologic changes and secure a biopsy and also because clinical experience has shown that roentgen findings are not final since about 10 per cent of cases returned with a diagnosis of carcinoma have been erroneous and from 10 to 20 per cent of cases supposedly negative have been found to have endoscopic evidence of disease. The esophageal bougie is mentioned only to be condemned. The treatment of carcinoma has been more or less of a palliative nature. Early neoplasms involving the cervical portions of the esophagus have given the best results from a surgical standpoint. Irradiation therapy gives temporary benefit. In their opinion, intubation is an important palliative measure that makes the patients more comfortable by preventing starvation and obviates the need for gastrostomy.

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46. Beck, J. C., and Guttman, M. R.: Carcinoma of the Esophagus, Ann. Otol., Rhin. & Laryng **41**:1183 (Dec.) 1932.

Abstracts from Current Literature

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CEREBELLAR ABSCESS. SYDNEY NUSSBAUM and GEORGE WEBB, Am. J. Dis. Child. **43**:147 (Jan.) 1932.

The author reports the case of a child aged 11, which is of value in that it indicates the wisdom of watchful waiting in cases of brain abscess until localization of the infected area has taken place. The case developed from otitis media on the left side; signs indicative of inflammation of the left lobe of the cerebellum gradually appeared, and later those of increased intracranial pressure. The interesting feature of the case is that the patient recovered without any apparent residual signs of damage to the central nervous system.

LEAVITT, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

HEARING SURVEY, PUBLIC SCHOOLS, ALBANY, N. Y. E. MARTIN FREUND, New York State J. Med. **32**:791 (July 1) 1932.

All pupils in and above the third grade in the Albany (N. Y.) schools were given hearing tests with the phono-audiometer (type 4 A, Westinghouse). The methods of testing are described. Children were retested with the type 2 A phono-audiometer. Retesting is important, as a large number of children showing defects on the first examination were afterward found to be normal.

Of 9,741 children examined, 339, or 3.5 per cent, showed defective hearing, and 168, or 1.7 per cent, showed borderline defects, giving a total of 507, or 5.2 per cent, with defects in hearing.

A history of bilateral head noises was reported by 1,259 children, and 1,244 children gave a history of previous earaches. In the majority of instances the same children reported both of these conditions.

Of 9,741 children examined, 2,852 gave a history of having had their tonsils and adenoids removed and 85 reported mastoidectomies. While about one third of all the children had had their tonsils and adenoids removed, the prevalence of enlarged and infected tonsils and adenoids was large in the defective group. Of 507, 222, or 40 per cent, showed enlarged tonsils; 132, or 25 per cent, had infected tonsils, and 18 per cent showed adenoid vegetations. Of 40 sixth grade children with defective hearing, 72 per cent had achievement tests below normal.

A thorough survey of hearing made by a part time otologist is recommended for all pupils in or above the third year.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

THEORY OF FISTULAR SYMPTOM OF THE LABYRINTH. K. WITTMAACK, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:115 (Aug. 23) 1932.

In animal experiments Wittmaack found that compression of the perilymph space through its indirect influence on the endolymph space regularly produced a nystagmus to the side involved in the experiment whereas direct pressure (contact) on the exterior wall of the utriculus above the fenestra ovalis regularly produced a nystagmus in the opposite direction. It is known that in cases of fistula the erosion of the bony semicircular canal may lie exclusively in the region of the perilymph or in the region of the endolymph. The author thinks that the regular fistular symptom characterized by nystagmus to the diseased side takes place in the same way as nystagmus through experimental compression of the perilymph space. Production of the reversed fistular symptom in his opinion takes place in the same way as the reversed nystagmus produced experimentally by direct pressure on the wall of the utriculus. Compression of the perilymph space

produces a simultaneous "circular" compression of the endolymph space of the utriculus, because the cochlear duct does not permit a sufficient escape of endolymph to compensate for the pressure effect. That a circular compression of the endolymph space of the utriculus provokes a nystagmus to the side being tested conforms to the author's tonus theory. This theory assumes that the specific excitation of the sensory organ of the inner ear consists in a hydrostatically transmitted increase of pressure in the sensory structures in the endolymph. This explanation conforms with the fact that the regular fistular nystagmus is usually a combined horizontal and rotatory nystagmus. When pressure is applied directly to any point on the exterior wall of the utriculus, the endolymph may escape through the endolymphatic duct into the endolymphatic sac because the duct is not pressed shut as in compression of the perilymph. This displacement of the endolymph, besides decreasing the pressure effect, produces a reaction in the sensory organs which results in a nystagmus to the opposite side. If the fistula is in the horizontal semicircular canal the displacement reaction must affect the crista of the vertical semicircular canal as well as that of the horizontal one, resulting in a horizontal-rotatory nystagmus. In accordance with this theory, the author thinks that the form of the nystagmus may, under certain circumstances, permit conclusions as to the anatomic conditions which exist.

EDITOR'S ABSTRACT.

POSTURE OF THE HEAD IN TUMORS OF THE POSTERIOR FOSSA. B. S. BEILIN, Ztschr. f. d. ges. Neurol. u. Psychiat. **138**:664 (Feb.) 1932.

A characteristic posture of the head in lesions of the posterior fossa has been known since the time of Batten (1903). He reported a case of cerebellar tumor in which the head was bent toward the shoulders and the face was turned to the side of the tumor. This he found was directly opposite to the posture assumed after removal of the cerebellum. Stewart and Holmes found that the posture of the head is often characteristic in intracerebellar and extracerebellar lesions. The head is bent toward the side of the focus so that the chin points in the opposite direction. This posture is neither constant nor pathognomonic, for they found it in tumors of the pons, mesencephalon and frontal lobe. Other authors do not consider the posture of the head of much value in diagnosis, since it is turned now one way, now another. They have offered various explanations: Cushing looked on it as a defense against pain; Wilson, as a symptom of partial decerebration, and Horsley, as a vestibular sign. Brain found a rotated cerebellar position of the head in lesions of the internal ear, the eighth nerve, the pons, the midbrain, the cerebellum and the forebrain. He concluded that this posture is a consequence of disturbance of a tonic reflex from the labyrinth under the influence of which the extensor muscles of the neck maintain equilibrium and overcome the weight of the head. Experimental destruction of one labyrinth or auditory nerve produces a rotation of the head to the injured side. Foci in the pons, midbrain or forebrain can produce a rotation, either to the side of the focus or to the opposite side. Foci limited to the cerebellum produce no rotation. There is rotation toward the diseased side in diseases of the middle ear and in tumors of the acoustic nerve. In five cases in which the pons and midbrain were involved there was rotation to the normal side. Cerebellar tumors may cause rotation either to the normal or to the diseased side.

Beilin studied the posture of the head in tumors of the cerebellopontile angle, tumors of the cerebellar hemispheres (extracerebellar and intracerebellar), tumors of the midline (vermis and fourth ventricle) and intrapontile tumors. There were twelve tumors of the cerebellopontile angle, all but one of which were tumors of the acoustic nerve. In all twelve cases there was a characteristic posture of the head: The chin was turned toward the side of the tumor; the ear was turned to the opposite side. In explanation of this, the following possibilities are suggested: 1. The spinal accessory nerve was irritated by the tumor and by overaction caused rotation of the head. 2. The posture of the head was compensatory for diplopia. 3. It arose as a disturbance in the tonus coming from the labyrinth and cerebellum.

The rôle of the spinal accessory nerve may be dismissed. While the head is rotated chiefly by the muscles innervated through the spinal accessory nerve, other muscles too are concerned, innervated through the cervical nerves. Hence, stimulation of the spinal accessory nerve alone is not enough to produce the posture of the head in question. Furthermore, in Beilin's cases of tumor of the cerebellopontile angle, the chin was directed toward the side of the tumor. This indicates a loss of function of the eleventh nerve, rather than an overstimulation. Further, the eleventh nerve is rarely involved in tumors of the cerebellopontile angle. The suggestion that the posture of the head is a compensation for diplopia is not logical. According to this theory, the patient assumes a certain position of the head in order to shut out the false images. If the muscles rotating the eyes to the left are paralyzed, the head is turned to the left; if there is paralysis of upward and downward movement, the head is pushed forward. The difficulty with this theory lies in the facts that postures of the head of the type described in this paper were observed in patients without diplopia, that closing of the eyes does not change the position of the head, and that such postures are seen in blind patients.

The labyrinth, therefore, plays an important rôle in postures of the head. The otolith apparatus has a direct influence on the postures of the head (Ewald), and any pathologic change in this apparatus is followed by a corresponding change in posture. Beilin attributes the posture of the head in the cases of tumor of the cerebellopontile angle to an irritation of the vestibular nerve. Bartels found a deviation of the head in rabbits after section of the eighth nerve, similar to the posture in the cases of tumor of the angle. After section of the right acoustic nerve the snout was turned to the left and vice versa. Similar results were observed by Leidler and van Gehuchten. After section of the vestibular nerve and the fibers from the vestibular nerve to the posterior longitudinal bundle, they found a deviation of the head toward the side of the focus; if the latter fibers were cut at the level of the genu of the facial nerve there was, in addition to the rotation of the head, a rotation of the body on its long axis.

Beilin was unable to find any constant posture of the head in his cases of cerebellar tumor. In extracerebellar tumors the posture of the head was similar to that in the tumors of the cerebellopontile angle. In cases of intracerebellar tumor, the posture of the head is exactly the opposite from that in tumors of the angle; that is, the chin is turned to the healthy side and the back of the head is toward the tumor.

In midline tumors of the posterior fossa the posture of the head is characteristic. Beilin's findings agree with those of Heuvers, that in cases of block between the third and fourth ventricles the head is flexed backward, but in cases of block between the fourth ventricle and the cisterna magna the head is bent forward in order to relieve the compression of the medulla.

In intrapontile tumors, the head may be turned either to the healthy or to the diseased side.

ALPERS, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

INTRACRANIAL COMPLICATIONS IN SIMPLE CHRONIC OTITIS MEDIA WITH CENTRAL PERFORATION. N. SADOWSKI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:590 (July 22) 1932.

Sadowski reports a case which falls into this rare category. The patient, a man, aged 39, with chronic otitis media of twenty years' duration, was hospitalized because of pain in the left ear and fever. As roentgenography revealed no softening of the mastoid, operation was declined. The patient exhibited a septicemic temperature curve and had daily chills; the blood culture revealed Streptococcus haemolyticus. At examination by the author six weeks after the onset of the illness, the left tympanum appeared slightly reddened and a small central perforation was seen. Through the perforation the reddened mucosa of the promontory could be seen. A few drops of nonfetid pus were seen in the meatus and on the floor of the tympanum. Antrotomy showed the mastoid antrum surrounded by a few cells containing no pus or cholesteatomatous mass. Further intervention revealed extensive thrombosis of the jugular vein; resection was performed. The

patient died six weeks after the operation following a general decline marked by continuation of the septicemic temperature curve and increasing chills. This case illustrates that in cases of septicemia in which there is a suggestion of intracranial complication more importance should be attached to the general condition than the reassuring finding of an apparently harmless chronic otitis media with central perforation. In the majority of cases of this kind pus is found in the epitympanum; this leads to necrosis of the tegmen tympani, pressure on the dura, perforation into the sinus and so on. In other cases, there is no retention of pus in the bone. Since not a drop of pus was found in the entire mastoid process and in view of the extensive thrombosis of the jugular vein, the author thinks that there was probably a primary thrombosis of the bulb of the jugular vein. The only pus found was on the floor of the tympanum. Several cases of direct perforation of the suppurative process from the floor of the tympanum in the direction of the bulb of the jugular vein with primary thrombosis of the bulb have been reported.

EDITOR'S ABSTRACT.

A CASE OF HEALED SUBACUTE OTITIS WITH SECONDARY LOCALIZED CEREBRAL SYMPTOMS. V. HLAVAČEK, Časop. lék. česk. **69**:1041 (July 11) 1930.

A boy aged 9 years, who had had scarlet fever and otitis media three years previously, which had lasted only several weeks, came to the hospital with a purulent otitis of three weeks' duration. There was an indurated, red, painful, but not fluctuating mass over the zygoma. The temperature was septic; the aural canal discharged thin, yellow pus, and the mastoid was tender. Antrotomy was performed with the patient under general anesthesia. A week later vomiting was followed by an evanescent urticarial rash and a slow response to questions. Two days later, epileptiform clonicotonic convulsions, facial palsy and coma appeared. Both the upper and the lower extremities were involved. There was a loss of abdominal, cremasteric and tendinous reflexes. Another operation revealed an inflammatory reaction about the root of the zygoma and a purulent membrane. The dura was tense. Puncture revealed clear fluid. Complete paralysis of the contralateral upper and lower extremities was followed by a slow recovery. Symptoms of this type may take place with increased intracranial pressure due to an extradural pathologic process, but they are not followed by paralysis and are only of short duration. The symptoms point to involvement of the hemisphere motor centers. A review of the literature is given, in which Korner's series are cited. Two cases occurring in the age group of from 4 to 7 years, eight cases in that from 10 to 15, four cases in that from 20 to 25, four cases in that from 30 to 38, and one case in a person 51 years of age are reported.

STULIK, Chicago. [AM. J. DIS. CHILD.]

Pharynx

A SMALL MILK-BORNE OUTBREAK OF SEPTIC SORE THROAT. ROBERT E. ARCHIBALD and ROY F. FEEMSTER, New England J. Med. **206**:1252 (June 16) 1932.

Twenty cases of septic sore throat were definitely traced to milk from a single cow. The rate of infection was high, 87 per cent of the persons definitely known to have used the milk raw becoming ill. There were three deaths, giving a fatality rate of 15 per cent. Two patients died of pneumonia and one of otitis media, cellulitis and septicemia. Hemolytic streptococci were found in cultures taken from the throats of fifteen of the patients and from that of the milker, and in milk from the diseased quarter of the cow's udder. No cases developed in contacts in the family not drinking the infected milk.

The usual clinical picture was that of a sudden onset with chilliness, fever and a dry, raw throat. There were also nausea, vomiting, backache and prostration.

Examination of the throat showed a bright red pharynx, uvula and soft palate, no exudate being visible. In all of the cases there were more or less swelling and tenderness of the cervical glands.

GENGENBACH, Denver. [AM. J. DIS. CHILD.]

A STUDY OF THE EFFECT OF SEVEN HUNDRED AND THIRTY-SIX TONSILLECTOMIES AND SEVEN HUNDRED AND FORTY-ONE CONTROLS. J. D. MONROE and V. L. VOLK, Rep. Am. Child. Health A. **6**:15, 1929.

In this study the following conclusions were drawn from the results of 736 tonsillectomies and the observations on 741 children who refused operations: 1. Greatest relief is obtained from sore throats, head colds, mouth-breathing and malnutrition. 2. Complaints were relieved in the first six months in 91 per cent of the cases. 3. Operation in early childhood offers the best results. 4. Forty per cent of the mentally subnormal or retarded children were improved. 5. Coexisting conditions, except probably pulmonary tuberculosis, are not contraindications for tonsillectomy. 6. The control group showed no definite improvement in a similar period of observation. Excellent tables are furnished.

PARKER, Rochester, N. Y. [AM. J. DIS. CHILD.]

SORE THROATS OF OTHER THAN TONSILLAR ORIGIN. BEDFORD RUSSELL, Brit. M. J. **2**:485 (Sept. 12) 1931.

The author refers briefly to the fact that in many instances operations on the tonsils are not complete and that remnants of tonsils are left. After a review of the physiology of the nose and throat, he points out that many times sore throats are due to recurrent sinusitis, with special reference to the antrum and with a secondary allusion to the ethmoid cells. A careful history, he states, in many instances will elicit the true cause of the recurrence of sore throat. He points out, by example and otherwise, that relatively young children, certainly as young as 3 years of age, have various forms of sinusitis. The sore throat in tonsillectomized patients is usually worse in the morning. Sometimes there are rashes on the skin of nose and lips, redness of one eye, pain or inflammation in one ear and an excess of secretion sticking to the hairs of one nostril. He emphasizes the fact that the discomfort is usually unilateral. Laryngeal symptoms and cough are more frequently evidence of sinusitis than they are of tonsillitis. The postnasal space should be examined carefully after the shrinkage of the nasal mucosa. Then a trickle of mucopus may be seen coming over the posterior end of the middle turbinate, which is presumptive evidence of posterior ethmoiditis or sphenoiditis.

The treatment is then discussed and is along relatively conventional lines.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

ACUTE TONSILLITIS AND SOME OF ITS SEQUELS: EPIDEMIOLOGICAL AND BACTERIOLOGICAL OBSERVATIONS. J. A. CLOVER and F. GRIFFITH, Brit. M. J. **2**:521 (Sept. 19) 1931.

Tonsillitis is considered a local manifestation of infection with hemolytic streptococci. The subsequent course of the infection is dependent on two factors, the pathogenic qualities of the infecting strain and the state of immunity of the person attacked. The site of the infecting dose is a contributory factor. It is strongly intimated in this article that, though tonsillitis and scarlatina are probably separate entities, nevertheless it is felt that the presence of the rash with the possible difference in the greater risk of nephritis makes the two one condition, both epidemiologically and bacteriologically. Infection is conveyed (1) by milk or cream, (2) by such vehicles as tableware, pencils, handkerchiefs and possibly by impure swimming-bath water and (3) by droplet infection.

In plate cultures from throat swabs taken from patients with acute tonsillitis, hemolytic streptococci are almost invariably found in profuse or even in pure culture.

There are three sequels of major importance to an attack of tonsillitis. These are scarlet fever, otitis media and acute rheumatism. The author feels definitely that scarlet fever may be referred to as the sequel to tonsillitis, quoting Trousseau's phrase: "Sore throat is an essential part of scarlet fever; it precedes the eruption."

The authors consider it as almost positive, although experimental data are lacking, that carriers are responsible for a large percentage of the outbreaks due to hemolytic streptococci. They also refer to *scarlatina sine eruption.* The bacteriologic study of scarlet fever in this article conforms more or less closely to the conventional studies already published by others. The same doubt apparently exists in the minds of the authors regarding the validity of the Dick reaction that exists in America today.

The troublesome nature and seriousness of otitis media are discussed. It is especially troublesome in large boarding schools. The experience of the authors in one boarding school showed a much higher incidence of otitis media among boys than among girls.

The troublesome question as to the relationship between the removal of tonsils and adenoids and subsequent infection of the middle ear arises in the minds of these authors, as it has arisen in the minds of most observers.

The question as to whether acute rheumatism is the sequel or complication of tonsillitis is likewise discussed, a number of authorities being quoted. It is felt that there is a definite relationship. In many instances there is the attack of tonsillitis that supplies the missing evidence in the previous history as to whether the patient has had rheumatism or not. The authors feel strongly, however, that epidemics of tonsillitis are frequently followed by severe relapses of acute rheumatism in children who have already had rheumatic treatment, and they draw the conclusion that children who have been subject to rheumatism should be protected as much as possible from any infection with the hemolytic streptococcus. Various suggestions have been made as to the care and the exercise to prevent outbreaks of acute tonsillitis.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

RIGIDITY OF NECK FOLLOWING ADENOTOMY. K. BERNFELD, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:293 (Sept. 21) 1932.

Bernfeld says that rigidity of the neck after adenotomy recently reported by Schaeffer as a rare complication is not so rare as the latter supposes. The author reports two cases. In both of them, as in Schaeffer's, there was no significant demonstrable glandular swelling. As malformation of the epipharynx existed in both cases, Bernfeld thinks that an injury of the deeper muscles incurred during intervention in the constricted nasopharynx resulted in myositis and consecutive rigidity of the neck.

EDITOR'S ABSTRACT.

CERVICAL ACTINOMYCOSIS: REPORT OF A CASE. A. NEUMANN, Monatschr. f. Ohrenh. **66**:910 (Aug.) 1932.

Following a description of the anatomicopathologic and the clinical picture of actinomycosis, Neumann reports a case of cervical actinomycosis of therapeutic interest. Three weeks after an attack of tonsillitis, a woman, aged 22, had a swelling on the left side of the neck which clinically appeared to be a glandular abscess. It receded quickly after incision and drainage. Two weeks later swelling reappeared at the site of the incision. A second incision revealed pus in which actinomycosis granules were demonstrated. Internal iodine medication was without result, and three weeks later there was a renewed abscess formation with a rapid spread downward. The abscess ruptured spontaneously, and antiphlogistic treatment was given. After abatement of the acute inflammatory symptoms, the diseased portion of the neck was resected. The resection was extended well into the healthy tissue. In the upper half the disease process extended to the superficial cervical fascia, but in the lower half the omohyoid and the sternothyroid muscles had to be resected. A few granules had to be removed from the surface of the

thyroid. The portal of entry for the infection was undetermined. The teeth were healthy. Nothing was observed on the tongue, tonsils, oral or pharyngeal mucosa which could be considered as the primary infection.

EDITOR'S ABSTRACT.

CERVICAL THROMBOPHLEBITIS AND SEPSIS SECONDARY TO TONSILLAR INFECTION. A. GREIFENSTEIN, Virchows Arch. f. path. Anat. **281**:748, 1931.

Greifenstein presents a study of four cases of tonsillar and peritonsillar infection that led to thrombophlebitis of the large veins of the neck and fatal sepsis. He compares with these one case of thrombophlebitis and sepsis following otitis media, one following the extraction of a tooth, and two following nasal furuncle. The tonsillar infection led to diffuse phlegmonous lymphangitis of the deep tissues of the neck, and this in turn to phlebitis and thrombosis of the large cervical veins, from which the infecting organisms entered the blood stream. Then venous involvement may lead to discrete intimal inflammatory lesions over which multiple discontinuous thrombi form. In the cases of sepsis following otitis media and extraction of a tooth, spread of the infection to the lungs was ascribed to propagation of the thrombophlebitic process into the collaterals of the jugular vein. In the two cases of nasal furuncle, the process was propagated along the veno angularis and vena ophthalmica to the cavernous sinus.

O. T. SCHULTZ. [ARCH. PATH.]

Larynx

LARYNGITIS SICCA GRAVIDARUM. E. POLLAK, Monatschr. f. Ohrenh. **66**:897 (Aug.) 1932.

Pollak states that not infrequently there develops during pregnancy a laryngitis sicca which by its resistance, spontaneous cure after parturition and relatively frequent recurrence in different pregnancies of the same person indicates an unmistakable relation to pregnancy. The laryngitis may occur in the first or in later pregnancies, and frequently recurs in several pregnancies. It may develop in the early or late stage of pregnancy, and in the majority of cases lasts until its termination. The termination of pregnancy brings about a marked subjective and objective improvement of the laryngeal condition, and soon after confinement hoarseness, cough and dyspnea usually disappear. Laryngoscopy a few months after confinement shows a complete change in the appearance of the laryngeal mucosa. The vocal cords present a normal appearance, without redness or secretion; their edges are sharp, and complete adduction is possible. The clinical picture of laryngitis sicca gravidarum is variable. In most cases it assumes the typical form characterized either by a too scanty secretion on the mucous membrane or by a tenacious secretion which dries in crusts on the laryngeal mucosa. In a small number of cases it assumes a hemorrhagic form. The pathologic changes almost always extend in slight degree to the tracheal mucosa, and in some cases the trachea and the larynx from the labium vocale downward are the main seat of the objective symptoms. In most cases this laryngitis occurs independently of existing analogous changes in superior parts of the respiratory tract. Its clinical significance lies in the fact that it is confused with tuberculosis of the larynx or associated with other forms of tuberculosis, especially pulmonary. When the crusts in the larynx are abundant, they may simulate a tuberculous ulcer. The not infrequent involvement of the posterior wall of the larynx also suggests tuberculosis. Stork has pointed out that the mucosa of the interarytenoid and the arytenoid region which frequently exhibits changes during pregnancy is also a favorite localization for laryngeal tuberculosis in women. For this reason it is often difficult in demonstrated cases of pulmonary tuberculosis to determine whether the laryngeal changes are due to pregnancy or to beginning laryngeal tuberculosis.

EDITOR'S ABSTRACT.

OLFACTORY REGION AFTER TOTAL EXTIRPATION OF LARYNX. A. HEINDL, Monatschr. f. Ohrenh. **66**:931 (Aug.) 1932.

Heindl's investigations of the sense of smell in persons who had undergone total extirpation of the larynx confirmed the observations of Ullmann. The latter found that such persons were wholly or almost wholly unable to smell odoriferous substances held in front of the nose or blown into the nose. He attributed this to destruction of the muscles extending from the floor of the mouth to the larynx and the sternum. When intact, these muscles enable the person to execute sucking movements with the pharynx by which air is sucked into the olfactory region even when breathing takes place entirely through a tracheotomy tube. As Ullmann found a great decrease in the sense of smell for odoriferous substances blown into the nose as well as those held before it, the explanation of atrophy of the sensory epithelium of the olfactory region due to disuse was indicated. The author examined histologically the olfactory mucosa of six patients who had died from nine and a half to seventeen months after total extirpation of the larynx. All of them had had a good sense of smell before exclusion of the nasal breathing. Degenerative changes were found in the sensory epithelium of the olfactory region, which confirmed the conception of atrophy. The changes were most pronounced in those who had exhibited the greatest decrease in the power of smell after the extirpation.

EDITOR'S ABSTRACT.

RESONANCE PHENOMENON OF THE SINGING VOICE. D. WEISS, Monatschr. f. Ohrenh. **66**:964 (Aug.) 1932.

Weiss has observed that when one sings into a glass tube the latter is set into vibration by some of the tones, but in the case of certain tones the tube not only does not vibrate but hinders voice production. This manifests itself in a wavering between two halftones or an inability to sing the note in question and the substitution of an adjacent halftone. These breaking points in resonance occur approximately in fifths. The author thinks that the explanation can at present only be guessed at. He says that the vocal organs constitute a soft-walled pipe which is able to act as a resonator for a wide variety of tones in approximately the same position; the glass tube adds a rigid component which is, perhaps, totally out of tune for some tones.

EDITOR'S ABSTRACT.

ANATOMY AND PATHOLOGY OF THE LARYNGEAL VENTRICLE. H. SCHRÖDER, Virchows Arch. f. path. Anat. **281**:330, 1931.

In one hundred fresh necropsy specimens, the size, length and contour of Morgagni's pouch of the larynx bore no relation to age or sex. Lymphoid tissue, although present in the wall, is not relatively prominent enough to warrant considering the pouch a lympho-epithelial organ. Enlargement of the reaction centers of the lymphoid follicles was frequently seen, but no relation between hyperplasia of the lyphoid tissue of the pouch and status lymphaticus was noted. Characteristic pathologic changes were seen only in tuberculosis and diphtheria. The multiplication of diphtheria bacilli in the pouch may lead to the carrier state.

O. T. SCHULTZ. [ARCH. PATH.]

CONGENITAL MALDEVELOPMENT OF THE LARYNX. ANNELISE ROHRMANN, Virchows Arch. f. path. Anat. **283**:304, 1932.

The anomaly occurred in a new-born girl with multiple malformations, and consisted in rudimentary development of the epiglottis. In place of the latter there was a group of cartilaginous nodules.

O. T. SCHULTZ. [ARCH. PATH.]

UNUSUAL THERAPEUTIC RESULT IN BILATERAL PARALYSIS OF VOCAL CORDS. D. WEISS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:634 (July 22) 1932.

Weiss reports the case of a woman, aged 29, who sought medical care three months after thyroidectomy because of complete aphonia due to bilateral paralysis of the vocal cords. After trying several customary therapeutic measures in vain, he requested the patient to do Fröschel's exercises (*Stossübungen*). These consist in energetically thrusting the arms downward while forcefully pronouncing a vowel or an explosive consonant with a vowel joined to it. In the course of these exercises, the right vocal cord, which was in intermediate position, gradually approached the other one, which remained unalterably in the paramedian position, until an almost complete glottic closure was brought about, whereby a surprising phonetic result was achieved. The author thinks that the case indicates the necessity for revising the present conception of intermediate (cadaver) position and paramedian (posticus) position.

EDITOR'S ABSTRACT.

Nose

ADENOIDITIS AS A PUBLIC HEALTH PROBLEM IN ITALY. A. M. CAYREL, Paris méd. **2**:283 (Oct. 8) 1932.

Adenoid infections may be divided into three groups: (1) vegetations which infect the ears and nose; (2) vegetations that are sufficient to produce rhinopharyngeal irritation, and (3) those sufficient to cause inflammatory nasal stenosis. Inhalations of bromo-iodide salts (obtained as a natural residue from Salsomaggiore water) are recommended. Patients in the first group are benefited by fifteen inhalations, those in the second and third, by thirty. The author examined 4,891 school children and found that from fifteen to twenty in a hundred had adenoiditis. After the aforementioned treatments had been given this number was decreased to two in a hundred who required operation.

SANFORD, Chicago. [AM. J. DIS. CHILD.]

RETROBULBAR NEURITIS OF NASAL ORIGIN. E. VON HIPPEL, Arch. f. Ophth. **128**:23 (Feb.) 1932.

The essence of this paper is von Hippel's reply to an acute question of the present day: Is the improvement of the retrobulbar process that frequently follows surgical procedures on the sinuses a post hoc or a propter hoc proposition? Are the "successes of the nasal therapy" actually the result of nasal operations, or are they produced by spontaneous recovery? Von Hippel believes the latter to be the case. He bases his conclusions on a new series of thirty-six cases (in addition to his first series of seventy cases), in thirty of which there was no pathologic condition in the sinuses on thorough roentgen examination. The remaining six patients whose sinuses were not investigated roentgenologically presented definite cases of multiple sclerosis or hereditary optic atrophy. Twenty-one of the thirty-six patients were cured; in ten the condition showed an unfavorable course, and in five the final outcome could not be determined. In the ten unfavorable cases operative procedures were, a priori, out of the question because of the underlying definite general pathologic conditions (multiple sclerosis, hereditary atrophy, myelitis). No patient was operated on. If operations had been performed in every case, the surgical method of treating retrobulbar neuritis would again have scored 70 per cent successes. The same consideration applies to the endonasal epinephrine tampon treatment. Hereditary optic atrophy is surprisingly frequent in von Hippel's material.

P. C. KRONFELD. [ARCH. OPHTH.]

MALFORMATION OF THE SKULL IN CASES SHOWING A NASO-ORBITAL ENCEPHALOCELE. H. MUSSGNUG, Frankfurt. Ztschr. f. Path. **42**:238, 1931.

Two cases are reported. In the first instance there was a defect of the skull involving the left frontal bone, and the ethmoid, right nasal and maxillary bones.

The left lacrimal bone was entirely missing. In the second instance, the ethmoid, maxillary, nasal and lacrimal bones were involved. The author believes that both conditions, the malformation of the skull and the encephalocele, were the result of one common cause, probably the effect of an outside force directed on the anlage of the skull early in the development of the fetus.

O. Saphir. [Arch. Path.]

Manometric Measurement of Air Exchange in the Maxillary Sinus and Its Practical Significance. K. Schmücker, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:638 (July 22) 1932.

Schmücker's measurements of the air pressure in the maxillary sinus confirmed the fact established by Braune and Clasen in 1877, that during inspiration through the nose there is a negative pressure in the maxillary sinus, while during expiration through the nose the pressure in the sinus is positive. This means that during inspiration air from the accessory nasal sinuses is mixed with the inspired air, and during expiration air from the nose enters the nasal sinuses. On the basis of present experience, the author thinks that measuring this air exchange by means of the pressure difference constitutes a simple method of completing an examination of the accessory nasal sinuses. After a customary puncture of the maxillary sinus from the lower nasal duct, a water manometer of glass is connected with the puncture cannula by means of a rubber tube. Pressure readings are taken during forced inspiration and expiration, with closing of the other nostril. It is essential to avoid getting a single drop of water or blood in the puncture cannula. If the pressure measurement in the maxillary sinus indicates a sufficient air exchange, if irrigation is unimpeded and if the irrigation fluid is clear, a disease of the sinus can be excluded with utmost probability. Injection of iodized poppy seed oil and roentgenography may be omitted in such cases. However, if an insufficient exchange of air is revealed by the pressure measurement, roentgenography is indicated to determine the existence of polyposis of the sinus even if the nasal examination and test irrigation gave negative results. The method is also valuable in demonstrating disturbances of air exchange in the maxillary sinus which cannot be diagnosed by any other method, but which play an important rôle in the development of headache.

Editor's Abstract.

Teeth in the Nose. H. Leicher, Ztschr. f. Laryng., Rhin. (Teil 1: Folia otolaryng.) **22**:445 (June) 1932.

Leicher reports four cases in which teeth were found in the nose. In most cases, these rare manifestations are due to inversion of a tooth, so that in place of the inverted tooth that has grown into the nose, there is a gap in the teeth. The first case was one of inversion of the tooth. Anamnesis at first indicated that it was caused by trauma at the age of 3½ years, but the stage of development of the permanent incisors at this age makes a turning of 150 degrees as a result of trauma improbable. Further, a sister of the patient also had a gap in the permanent teeth without a history of trauma. Roentgenography in her case indicated that the missing incisor was inverted in the hard palate. This was probably a case of congenital anomaly of development. The second case concerns a woman with a supernumerary tooth. The third case concerns a boy, aged 8, with a cleft palate. Operation on the cleft palate disclosed the root of a tooth which extended about 4 mm. into the nose. In the fourth case, the tooth did not grow into the nose, but merely lodged there. Presumably it was pushed into the maxillary sinus during attempted extraction, where it caused suppuration. In the course of irrigation of the sinus, the tooth arrived in the nose through a large opening in the lateral nasal wall.

Editor's Abstract.

TISSUE STRUCTURE AND DEVELOPMENT OF MUCOSA OF ACCESSORY NASAL SINUSES AND ITS INDIVIDUAL NATURE IN COMPARISON WITH MUCOSA OF MIDDLE EAR. M. SCHWARZ, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:459 (June) 1932.

In 1931, Schwarz reported that in a study of the mucosa of the middle ear on one hundred fetuses he found significant individual differences in the thickness of the layers of the mucosa as well as in its structure. This variability was found to be due to the nature of the constituent elements of the connective tissue, especially the fibroblasts and the collagenous fibers. The fibroblasts of the mucosa of the middle ear develop with individually varying rapidity from the young forms to the long connective tissue cells. The tendency to fibril formation is also individually variable. The author has studied the mucosa of the accessory nasal sinuses to decide whether this individually variable nature of the constituent elements of the connective tissue also affects other mucous membranes, whether this local individuality is a manifestation of a universal variability of the connective tissue, and, further, whether the intra-uterine development of the mucosa of the middle ear parallels that of the nasal sinuses. It was found that the latter is not the case. In the nasal sinuses loose fibrillar connective tissue is seen in a stage when embryonic connective tissue is usually still found in the middle ear. Furthermore, the matrix of the connective tissue shows variations not only in different sinuses, but in different parts of the same sinus. Despite these complicating factors, the individually variable tendencies of development of the fibroblasts are unmistakable. The same is true of the infiltration of collagenous fibers. No mucosa is identical with another mucosa in the finer details of tissue morphology. A comparison of the mucosae of the different nasal sinuses with each other and with the mucosa of the middle ear in the same patient showed that uniformity is the rule. Uniformity of the fibroblasts was observed in 90 per cent of the cases and of the collagenous fibers in 94 per cent of the cases. This uniformity in different persons, despite great variations, indicates the probability that the fibroblasts and the collagenous fibrils of the entire organism are of a uniform nature.

EDITOR'S ABSTRACT.

CONSERVATIVE OPERATION ON THE MAXILLARY SINUS: RELATION TO THE TEETH. S. UNTERBERGER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**: 466 (June) 1932.

Unterberger thinks that the present methods of testing for electrical excitability by faradic and galvanic current are insufficient to establish whether or not impairment of the sensitivity of the teeth follows a radical operation on the maxillary sinus. The reliability of the results of the test obtained with high frequency current has not yet been established. Since it has not been proved that the radical operation does not injure the teeth, the author thinks that its use should be limited. He recommends a conservative endonasal method of operation on the maxillary sinus, avoiding the facial route and preserving the involved mucosa. He describes his modification of Halle's method, which has for its aim the preservation of the teeth. It simplifies Halle's technic and is more suited to the purpose, since it permits a view of the region of the natural opening of the maxillary sinus, where the severest changes of the mucosa are often located, and removal of these if necessary. It is preferable to radical operation in mild cases, in those in which the chief changes in the mucosa are in the region of the median wall of the maxillary sinus, and also in cases of genuine polyposis of the sinus, when the condition is not multiple, but is in the nature of a more isolated polypous development. The method is particularly suited to antrochoanal polypi. In cases of severe suppurative disease of the maxillary sinus affecting the entire mucosa, one can either combine the radical operation immediately with the endonasal intervention described, or try to bring about healing by irrigation therapy and undertake radical operation only if this fails.

EDITOR'S ABSTRACT.

INHALATION OF NEGATIVELY IONIZED AIR IN NASAL AND AURAL DISEASE. H. LEICHER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:497 (June) 1932.

Leicher reports good results with the inhalation of negatively ionized air in several diseases of the nose and ear. While the value of this therapy in nasal and aural disease cannot be finally established because of the small number of cases observed, it may be said that in some forms of tinnitus aurium and vertigo, in certain cases of vasomotor rhinitis, in some forms of chronic suppuration of the accessory nasal sinuses and especially in chronic purulent rhinitis this therapy is productive of results that cannot be achieved by any other method. Dessauer's apparatus for the production of negatively ionized air was used by the author. A series of twenty inhalations at from one to two day intervals is recommended. The inhalation periods may be gradually increased from ten minutes at first to thirty or even forty-five minutes. In cases of rhinitis sicca, pharyngitis sicca and laryngitis sicca, the influence of the inhalation was unfavorable.

EDITOR'S ABSTRACT.

Miscellaneous

THE HANDICAP OF CLEFT-PALATE SPEECH. DOROTHY M. WOLDSTAD, Ment. Hyg. **16**:281 (April) 1932.

An unrelieved cleft palate takes a human being out of contact with his own world. Filled with a sense of his inability to meet the social situation, he has feelings of inferiority and insecurity. This is an unnecessary tragedy; surgical skill is fully equal to the task of correcting the deformity, and pedagogy is entirely able to retrain the patient. The intelligence of children with cleft palate is within the normal range of intelligence quotients, and offers no obstacle to speech training. The author suggests that every obstetrician should examine the mouth of each child he delivers, and that babies who present this defect should be referred to the plastic surgeon within a month after birth. Correction at this early stage is surgically more simple than a later operation, and it has the additional advantage of saving the child from the emotional hazard of realizing that he has abnormal speech. Speech training must supplement surgical correction in older children. One of the major aims of this is to teach the child to project the air stream out of the mouth instead of through the nose; he should be taught resonance and tone quality. An effort should be made to qualify him in some other field, athletics or music, for example, so that he may have reason to feel superior as a balance to any feeling of inadequacy his defective speech may produce.

DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

SPEECH DEFECTS APPARENTLY CONGENITAL IN ORIGIN. I. M. ALLEN, Brit. J. Child. Dis. **29**:98 (April-June) 1932.

In part the author concludes that delay in learning to speak and defects in speech may be produced by disturbances of hearing or of the ability to appreciate the meaning of word sounds, by conditions leading to defective mental development or by conditions that prevent the development of normal control over the muscles of articulation. Psychologic situations can produce defects which may be confused, on a superficial examination, with those due to gross organic defects.

The exact nature of the defect in speech can be rarely determined from the study of the speech itself.

LANGMANN, New York. [AM. J. DIS. CHILD.]

LESION OF THE FOUR LAST CRANIAL NERVES ON THE LEFT SIDE. A. BACELAR, Rev. Assoc. paulista de med. **1**:280 (April) 1932.

Bacelar reports the case history of a woman wounded by a firearm. The bullet had entered the left canine fossa and traveled in an anteroposterior direction with-

out penetrating the buccal or pharyngeal cavities; it was arrested in the soft tissues of the neck behind the posterior border of the mastoid. The patient presented (1) left-sided paralysis of the tongue, the soft palate, the constrictor muscles of the pharynx, the larynx and the sternocleidomastoid and trapezius muscles; (2) loss of taste in the posterior part of the left side of the tongue and loss of feeling in the soft palate, the pharynx and the larynx, and (3) abundant pharyngeal secretion and tachycardia up to 140. Consequently the injury involved the glossopharyngeus, vagus, accessorius and hypoglossus nerves. Fifteen days later a second examination disclosed the same symptoms, except that the pulse rate had dropped to 110 F. One month and a half after receiving the injury the patient complained of loss of strength in the left arm, stating that it felt as if it were asleep. Reexamination showed marked atrophy of the tongue and the sternocleidomastoid and trapezius muscles, and the existence of an aneurysm 1 cm. behind the mastoid process, proving that the internal carotid had been injured. This aneurysm was the probable cause of the disturbance in the patient's arm from pressure on the brachial plexus. The patient disappeared from observation after she left the hospital.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

TRANSITORY CROSSED ABSENCE OF ROTATORY AND CALORIC REACTION IN A CHILD. E. VERMES, Monatschr. f. Ohrenh. **66**:689 (June) 1932.

Vermes reports the case of a child aged 7 who sustained a slight trauma of the skull. Two days later pains in the throat and ears appeared, and two days after that vomiting and vertigo set in, followed by facial paralysis. Examination in the otologic clinic revealed that all three branches of the right seventh cranial nerve were paralyzed, and that the fifth and ninth cranial nerves on the right side were also involved. The eighth cranial nerve was involved on both sides. On the right the rotatory reflex was lacking; there were slight underexcitability for irrigation with cold water, spontaneous nystagmus of the first degree to the left and a slight decrease in the hearing capacity compared to the normal hearing of the other side. On the left side there were vestibular nonexcitability for irrigation with cold water and normal excitability to rotation. Roentgenologically there were signs of great increase of intracranial pressure, but clinically there was no support for this. With the exception of a slight caloric underexcitability on the right side, the patient exhibited complete spontaneous recovery after two and one-half months. The pathogenesis is undetermined. The author considers the trauma too slight to be the etiologic agent; a cerebral polyneuritis of infectious toxic origin would be more probable. Other possible causes of the symptoms are mentioned.

EDITOR'S ABSTRACT.

ANALYSIS OF INVERSION OF EXPERIMENTAL OPTIC NYSTAGMUS. T. D. DÉMÉTRIADES, Monatschr. f. Ohrenh. **66**:809 (July) 1932.

Démétriades reports his analytic study of inversion of experimental optic nystagmus. In certain cases of spontaneous nystagmus there occurs, in the test with Brunner's revolving screen, instead of the typical experimental optic nystagmus in opposite direction to the rotation of the screen, a nystagmus in the same direction as the rotation of the screen; this is termed inversion. The author studied the relation between experimental labyrinthine nystagmus and experimental optic nystagmus by using the revolving screen and the revolving chair simultaneously. Experiments with the screen and chair turning in the same direction at the same and at varying speeds showed that the stronger nystagmus can overcome and neutralize the weaker one. When the screen and chair were rotated in opposite directions at the same and at varying speeds, it was shown that the slow component of experimental optic nystagmus is a stronger element than the rapid component when it is a question of influencing a previously or simultaneously existing nystagmus. When the rapid component of the experimental optic nystagmus occurs simultaneously with another concurrently existing nystagmus in the same direction, the

latter is reenforced. The experiments confirm the clinical experience that in cases of horizontal spontaneous nystagmus (optic nystagmus) there is an influence on experimental optic nystagmus. The direction of the experimental optic nystagmus is in the direction of the stronger summation. The author concludes that in testing spontaneous optic nystagmus with the revolving screen, the typical experimental optic nystagmus cannot occur for dynamic reasons, and inversion occurs in its stead. For the same reasons, the inversion, in some cases, is to be found only on one side.

EDITOR'S ABSTRACT.

COMPARATIVE STUDIES OF ANATOMIC AND PHYSIOLOGIC BASIS OF INSPIRATORY PHONATION. G. KELEMEN, Monatschr. f. Ohrenh. **66**:953 (Aug.) 1932.

Kelemen says that the inspiratory voice is unusually widespread in the animal kingdom, and that there is a wide variety of arrangements for its production. These reach a considerable degree of development in the highest form of animal life, the ape, and must be regarded as an enrichment of the entire voice production. Differentiation of the parts of the laryngeal lumen below the point of voice production can lead to an actual inversion of the entire phonetic arrangement of the larynx. The superiority of the human voice is due not only to man's oropharynx, capable of maximum modulation, his vocal cords, constructed with a degree of uniformity not found elsewhere, and the smooth funnel-shaped tube below the vocal cords, but to the uniformly expiratory phonation. The orangutan, possessed of an exceptionally differentiated thyro-arytenoid muscle, lost the ability to produce finely modulated tones through later development of the edge of the vocal fold which has no contact with the musculature. In an earlier stage of development frequent use of the inspiratory voice was an enrichment of phonation, but the author thinks that it may be regarded as a habit which had to be discarded in order that the most highly developed voice, the human voice, might be attained.

EDITOR'S ABSTRACT.

SURGICAL DIATHERMY IN OTORHINOLARYNGOLOGY: NEW INSTRUMENTS. M. KINDERVATER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:627 (July 22) 1932.

Kindervater recommends the use of surgical diathermy in the form of electrocoagulation in lupus and tuberculosis of the mouth, nose and pharynx, and in benign and malignant tumors for the prevention of hemorrhage and dissemination. For lupus he uses the button electrode. In a case of tuberculosis of the larynx he used a specially constructed diathermic snare for removal of the epiglottis, and for a tumor of the epipharynx he used a diathermic curet. In the larynx he does not use coagulation, because the depth of the action is difficult to determine. Several new instruments are described.

EDITOR'S ABSTRACT.

Society Transactions

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Annual Meeting, Atlantic City, N. J., May 23-25, 1932

LYMAN RICHARDS, M.D., Boston, *Abstractor*

END-RESULTS OF ONE HUNDRED CONSECUTIVE SINUS OPERATIONS. DR. HAROLD TOBEY, Boston.

One hundred patients with sinus disease were examined with respect to the value of various surgical procedures and the results obtained. These were listed in terms of relief to the patient's symptoms, not with regard to entire cure of the pathologic process, since one may exist without the other. Consideration was given to the age of the patient, the chief complaint, the objective findings and surgical treatment and the end-results, which sometimes had to be estimated since regions like the sinuses are liable to reinfection and, unlike the appendix, cannot be entirely removed.

The radical operations on the antrum were done in the Caldwell-Luc manner, with complete removal of the lining mucosa in all cases. All radical operations on the frontal sinus were done by the Lynch method, while ethmoidectomies were done by the intranasal or transantral route.

The youngest patient was 4 years of age and the oldest 73, the average being 34.

Chief complaints were divided between nasal discharge or obstruction, recurrent colds and recurrent frontal headache, the latter comprising two thirds of all complaints. Pus or nasal polypi were the commonest objective findings.

There were in all sixty-five radical operations on the antrum, fifty-eight ethmoidectomies, eight radical operations on the frontal sinus and fourteen intranasal operations on the antrum, either single or bilateral. Cure was obtained in 77 per cent of the sixty-five radical operations on the antrum and in 57 per cent of the fourteen intranasal operations on the antrum; cure was also recorded in 80 per cent of the six ethmoidectomies, in 75 per cent of the eight radical operations on the frontal sinus and in 50 per cent of the intranasal operations on the frontal sinus. With respect to the cure of symptoms, nasal discharge and obstruction were reported as cured in 77 per cent of the cases, recurrent colds in 80 per cent, recurrent sinus infection in 88 per cent and discharge following tooth extraction in 100 per cent.

Adequate hospitalization is necessary in the treatment in these cases, a radical operation on the antrum, for instance, requiring between seven and ten days. For most of the patients, the less after-treatment the better. An annoying complicaion is the occasional recurring edema about the scar and eyelid following radical operation on the frontal sinus. This is best handled by probing the nasofrontal duct, rarely by incising the previous scar. Edema of the cheek following radical operation on the antrum is relatively uncommon.

THE THEORY OF FOCAL INFECTION: APPLICATION. DR. EDWIN MCGINNIS, Chicago.

There was considerable ignorance in regard to focal infections in the early days of surgery, until the experiments of Billings and Rosenau in which streptococci were sealed into a tooth and rheumatic fever resulted. The same thing is now known to occur following human tooth infection and following infection in the tonsils. The commonest primary foci of infection are the tonsils, adenoids, teeth and sinuses, while listed as secondary foci are the gallbladder, appendix, prostate, cervix, lung and joints. Any chronic infection in the body may act as a focus. The teeth are a particularly popular focus due to unclean dental work

or infection from fillings or infection by the blood stream from other places. All tonsils may be regarded as infected, and only complete removal insures against complications such as rheumatism, endocarditis and similar diseases. In the infant, the adenoid is of particular importance as the source of infection in sinus and eustachian tubes. The sinuses themselves may act as foci, harboring bacteria in the lining membrane. The smaller the sinus the more the infection, since swelling closes the ostium and pushes bacteria into circulation. It is impossible to remove the entire membrane of all the sinuses. Some surgeons are thought to be too radical and some too conservative in this matter. Only by group practice can satisfactory diagnostic arrangements be made to cover all aspects of focal infection such as the x-ray, laboratory work, bacteriologic studies, examinations of the blood and physical examinations. A general conference must assemble all this data, and even then different opinions will result. End-results must be studied, a large number of cases considered, and decision made as to what foci are the most important to remove.

CARCINOMA OF THE LARYNX: CLINICAL AND PATHOLOGIC ASPECTS. DR. LOUIS H. CLERF, Philadelphia.

Early diagnosis of cancer of the larynx and complete removal of the disease are important. The larynx is the site of 4 per cent of all malignant tumors, 98 per cent of which are of the squamous cell type. Between a quarter and a third of these are seen early enough to be treated surgically, but unfortunately many are seen only from a medical standpoint and are not diagnosed. Examination with a mirror must be made, and it must be realized that chronic inflammatory lesions may be precancerous. Some of these will clear up with vocal rest, and it is hard to tell which must be followed more carefully.

A positive Wassermann reaction may give a false clue and lead to loss of time. If prompt improvement in these cases does not follow, biopsy should be done, since both conditions may be present to the extent of 4.5 per cent of combined syphilis and cancer in two hundred cases. Malignancy must be differentiated from tuberculosis, which in a typical case with pulmonary tuberculosis is an easy matter. In atypical cases, biopsy should be done, and again the two lesions may occur simultaneously. In each case no harm will result from the biopsy. Likewise, cancer may occur in the presence of pulmonary tuberculosis.

With respect to biopsy, there is some disagreement as to dangers from dissemination and the failure to check with clinical diagnosis. The pathologist cannot report on the growth in toto, but lack of proper technic or inexperience on the part of the pathologist may be the chief source of the trouble. If possible, the growth should contain an edge of normal tissue, and the base of the lesion must be secured. If the report is negative and the clinical findings suggestive, a second specimen should be taken. In early cases, it is sometimes hard to differentiate inflammatory lesions from cancer, and a frozen section method is not as satisfactory as a rapid paraffin process in eighteen hours.

Dissemination is likely only in a closed lesion and not in an open one such as is found in the larynx; there is no evidence to support the idea of laryngeal biopsy as resulting in dissemination. Biopsy on inoperable cancer has not shown this to be present more than in cases in which biopsy is not performed.

The histologic grading by Broder's method is a great advance in laryngeal work and demonstrates two extremes, low grade malignant squamous cell carcinoma with considerable differentiation of the infiltrating epithelium with keratin and a high grade anaplastic undifferentiated growth with a great variety of size and shape of cells and nuclei and many mitotic figures. The latter growth is only a small percentage of the total. Finally, there is a large intermediary group which may or may not be able to be subdivided. The cell type must be considered in conjunction with all other factors.

The fortunate fact that early metastases from laryngeal cancer are rare is a factor of great importance. Metastases to the lymph nodes occur very late in spite of the rich lymphatic supply of the larynx. The lower the grade of the cancer, the less metastasis. This fact should encourage radical surgical measures.

The type of operative attack must be determined by the location and extent of the growth. This must be based on mirror examination, prompt laryngoscopic examination and palpation of the neck. The degree of malignancy must be determined according to present microscopic standards and some form of radical surgical intervention such as laryngofissure or laryngectomy adopted. Endolaryngeal methods have no value. There are apparently no ill effects from preliminary biopsy. In the present series there were six cases reported as of low grade malignancy, two as of intermediate degree and six as anaplastic. Of the six recurrences, five were intermediate and one anaplastic.

BIOCHEMICAL CONSIDERATIONS IN OTOLARYNGOLOGY: THE PALE SEPTUM. DR. IRA FRANK, Chicago.

While the details of physical and chemical processes are of little practical value in daily health and disease, nevertheless, disturbance of the acid-alkaline equilibrium may be associated with many disease syndromes. Many patients exhibit a great variety of complaints without any definitely demonstrable lesions. Often a nutritional imbalance lies at the bottom of these cases, and there has heretofore been no ready method of discovering this faulty acid-alkaline balance. A rapid test is apparently to be found in the mucous membrane on the cartilaginous portion of the septum. In this connection, foods are best classified as producing an acid-ash and an alkaline-ash instead of dividing them into the typical proteins, carbohydrates and fats. It is in this connection that Jarvis has correlated diet and symptoms of the respiratory tract in that the change from an acid-ash to an alkaline-ash diet has seemed to modify the respiratory symptoms. Foods ripened by the sun are in general alkaline-ash producers, while carbohydrates and proteins are acid-ash producers. An excessive acid-ash diet gives a nutritional disaster syndrome of fatigue, irritability and anorexia, day-time listlessness, constipation and a dry skin. The septum under these circumstances is crimson red. Patients subsisting on an excessive alkaline diet are apt to exhibit allergic symptoms and to be subject to colds, hay fever and asthma.

Vasomotor rhinitis is a blood alkalosis characterized by a pale septum resulting apparently from the effect of the food on capillary permeability. High protein diet gives a red septum as a result of dilatation of the peripheral capillaries.

Thus, the nose is a valuable help in estimating the acid-alkaline balance.

Treatment is obvious, too much acid needing to be combated by the giving of alkaline-ash foods and soda, too much alkali by the administration of acid-ash foods and some mineral acid.

Naturally, purely local conditions and infections must first be ruled out, and methods previously proved satisfactory must be adhered to. Apparently the pale septum is more commonly seen than the red, perhaps because it produces more discomfort and hence sends more patients for treatment.

NONLIGATION OF THE INTERNAL JUGULAR VEIN IN THE TREATMENT OF SIGMOID SINUS THROMBOSIS. DR. O. JASON DIXON, Kansas City, Mo.

The classic procedure of ligation of the internal jugular vein dates from 1880 and is based on the false assumption that ligation of a vein arrests the progress of a phlebitis. Sigmoid sinus thrombosis is a defense mechanism in the control of spreading infection. If the occluding thrombus did not itself break down with the liberation of bacteria, ligation would be definitely contraindicated, and it is well known that thrombosis of itself may control an infection without surgical intervention. Even after the most radical surgical procedures, we often depend on the formation of a new and uninfected thrombus. Pelvic thrombosis has been found not to justify surgical intervention. I have felt that ligation of the internal jugular was not rational and that in cases in which it was omitted the patients have done as well or better without the added procedure.

The idea that the thrombus extends toward the heart is incorrect, this procedure taking place only until occlusion is complete. After this, it extends in the opposite direction, toward the head, as proved by injections of calcium chloride into a

cow's jugular vein. This extension to the head can be prevented only by removal of the thrombus during its formation, since the torcular end cannot be ligated.

The first pathologic stage is infection of the overlying bone by direct extension from infected mastoid cells, thus producing phlebitis. This first stage can be diagnosed, and if the overlying bone is widely removed, infection can be stopped. Other causes of the same symptoms must, however, be first ruled out. In this first stage one could do a complete exenteration of the mastoid and remove the inner table of bone until healthy sinus wall is exposed. Light packing of gauze treated with petrolatum is inserted without removal of the parasinus granulations, and the wound left wide open. If symptoms continue, it is easy again to expose the sinus, open it, and remove the clot.

In long-standing cases, the sinus may be covered with granulations or the wall of the vein may look red and lusterless, in which case an occluding thrombus has probably already formed. Once the endothelium is injured, platelets are deposited and a mural thrombus forms, going on to complete occlusion. Initial symptoms may subside but will probably recur. The use of a curet to remove the thrombus may cause trauma, and an extension of the infection and application of suction through a catheter are more efficacious. If extension has taken place into the bulb, it may be impossible to get free bleeding from below, but ligation even above the facial still leaves unligated tributaries such as the inferior petrosal which will carry infection sometimes to the cavernous sinus. It is better to place a wick in the bulb and give attention to the patient's general condition.

The argument that ligation prevents pulmonary embolism is false, because this may occur at the outset of the disease. If complete occlusion of the vein has occurred, there will be no emboli except by retrograde dissemination into the cavernous sinus or opposite jugular.

Other work has shown that pulmonary infarction cannot be prevented even by bilateral jugular ligation, and if sepsis continues after removal of the thrombus, then attention must be directed to general supportive measures and invidual resistance must be raised by good nursing, forcing of fluids and transfusion, the most important adjuncts in any fight against sepsis.

THE ASSOCIATION OF ACUTE SINUSITIS AND ACUTE PURULENT OTITIS MEDIA IN INFANTS AND CHILDREN. DR. EDWARD H. CAMPBELL, Philadelphia.

The lack of popular appreciation of the frequency of sinus infection in infants and young children is the underlying cause of other systemic diseases. That there is a close relationship between sinus infection and middle ear disease was shown by Fowler, in 1929, when he demonstrated an 86 per cent sinus infection in a series of one hundred cases of otitis media. Most children have had colds before reaching 1 year of age and continue to have them more frequently than adults. Practically all colds in infants are really instances of acute sinusitis due to extension of the nasal infection into the sinus. The infant's ostium is relatively larger than the adult's and sneezing, coughing, crying and vomiting cause regurgitation of secretions into the sinus.

The cause and persistence of infection in the infant lies in the lack of removal of the nasal secretions. It is the forcing of these into the eustachian tube that is the cause of the otitis media, since in the presence of sinus drainage the membrane of the swollen tube gives poor drainage to the middle ear and otitis media develops. In the absence of sinus infection, the tube is not swollen and drainage will take place.

A series of one hundred and fifty hospital patients was examined for this relationship. Gross inspection of the nose was undertaken as to the appearance of the nasal mucosa and amount and character of secretions; 2 per cent solution of cocaine was sprayed over the turbinates, and the nasopharyngoscope was used to determine the location of pus. The ears and throat were examined and roentgenograms were made in selected cases, the whole examination being easy and feasible, even in small infants. The use of hand suction is helpful in removing secretions and drawing them out of the sinus. Location of pus is a clue to which

sinus is involved, and this type of nasal examination is often more satisfactory than a roentgen examination.

Infants are bound to get infections of the upper respiratory tract which usually means sinus infections and then middle ear infections. The most practical treatment is suction. Suction bulbs can be placed in every ward and frequently used during the day by nurses, tending thus to lessen the incidence of otitis media. Organic silver salts are useless unless put directly into the sinus, and even here bacteria have been shown to be buried in the submucosal tissues. The hand suction bulb may be applied tightly to one nostril and a small amount of air allowed to enter through the other. Middle ear drainage may even be facilitated in this way, and fluid dropped into the ear canal through a perforation can be seen in the nasal passages.

The ethmoids seem to be more frequently involved, but often all sinuses are infected, especially in infants. A sinus and a contralateral ear may be involved. In gastro-intestinal disturbance, the sinus may be a more important focus of infection than the ear and its treatment more essential. The younger the child, the more common the infection. If sinus infections are properly handled, incision of the drum would not be necessary in many cases of otitis media.

Suggested Modifications of the Laryngofissure Operation. Dr. John M. Loré, New York.

This anatomic study is based on the importance of an adequate exposure of the larynx in the laryngofissure operation and on a study of forty cadavers. The key structure is the thyrohyoid membrane which runs from the upper border of the thyroid cartilage to the hyoid bone. It is pierced posteriorly by the superior laryngeal nerve and the superior thyroid artery and vein. A straight line drawn across at the level of the lamina of the thyroid cartilage to a point within 1 cm. of the superior cornu of this cartilage will localize the point of entry. The base of the epiglottis is also in relation to these same structures. Incisions in the thyrohyoid membrane to avoid these structures and vessels must start in the midline and go laterally and posteriorly, but anterior to the aforementioned line.

Anesthesia may be local or general, but the interior of the larynx must be cocainized. The field is then blocked with procaine hydrochloride, but if general anesthesia is preferred, intratracheal insufflation of anesthesia through a tracheotomy tube or even rectal anesthesia may be used.

If preliminary tracheotomy is performed, two incisions are used instead of one long one, and may be done at separated times. A thyrotomy skin incision is made from the body of the hyoid down to the upper border of the cricoid without skeletonizing the larynx. The cartilage can be divided with the Clerf saw without cutting the anterior commissure. The larynx is then opened by incising through soft structures in the anterior commissure. Here the usual difficulty in retracting the cartilages can be avoided by a second cut through the thyrohyoid membrane laterally and posteriorly but not including the skin. Thus a straight incision is changed to a Y-shaped one with the V part of the Y in the thyrohyoid membrane. This greatly increases the ease of exposure of the interior of the larynx and gives a wider aperture through which to work. Packing in the trachea prevents secretion from entering the larynx. The usual technic is employed in removal of the growth. A feeding tube is introduced into the nose, and bleeding vessels are ligated. No sutures are needed in the thyrohyoid membrane, and muscles and fascia are then approximated. Feeding should be carried out through the tube if necessary, and the tracheotomy tube removed as soon as possible.

Biochemical Considerations in Otolaryngology: The Red Septum. Dr. D. C. Jarvis, Barre, Vt.

The upper respiratory tract has been in many ways a useful guide to the state of general bodily health. Particularly the color of the nasal mucous membrane covering the turbinates and septum has been found to be indicative of varying states of bodily nutrition. It has been observed that the mucosa of the nasal

septum may be subject to four varying degrees of redness which may be correlated with certain variations in the clinical picture. It is often important to decide whether any given form of treatment should be directed toward the production of an acid or alkaline metabolic reaction. In this connection there are three important nasal factors. First, the color of the mucosa of the septum, second, the character of the nasal discharge and third, the presence of an associated, so-called nasal septum syndrome.

Four degrees of redness in the septum are detectable, and all indicate that the patient is selecting more food from the acid-ash-producing side than from the alkaline-ash-producing side, which can be proved by altering the proportion of foods from the standpoint of ash production and observing the changes in the degree of septal redness. In the same way, a pale septum will become redder on the reversal of this process. If nasal discharge is also present, it is an indication of a crossing of the individual line of tolerance for starches if it is mucopurulent and for sweets if it is watery.

Together with increased septal redness is an associated syndrome of fatigue, irritability, constipation and dry skin. These persons are particularly subject to acute infections and have little reserve energy.

Factors concerned with the production of a red septum are excessive acid-ash food, long continued intake of medicinal acids and fatigue due to an excessive output of muscular energy.

Treatment consists in having the patient select three times as many alkaline-ash-producing foods as acid until the septal color improves. Next, a teaspoonful of soda is taken three times a day for three days, then a glass of lemonade for three days, then soda for one day and lemonade for three days, alternating for a month. In addition, one teaspoonful of cod liver oil twice a day is taken until sleep is normally restful. In addition, a glass of orange juice at one meal and other fruit juice at the other two will be essential. Finally, a glass of water every hour all day long should be taken, to be reduced as improvement follows.

All this is important in discovering a patient who may otherwise show little on examination and yet who fails to respond to ordinary medical or surgical measures. This form of treatment does not supplant the usual procedures for combating infection but renders the bodily tissues less favorable soil for the growth of micro-organisms.

ROENTGEN DIAGNOSIS VERSUS CLINICAL DIAGNOSIS. DR. BERNARD P. WIDMANN, Philadelphia.

Clinical and roentgen diagnoses are not competitive procedures, but both are essential and mutually supplementary. The patient's problem is clinical, and often the clinical diagnosis is obvious and roentgenograms are only confirmatory and of use in a borderline case. In such, they may reveal pathologic processes not shown by clinical findings, though negative findings are not always conclusive. By roentgen examination it is sometimes hard to tell the difference between a healed process and a chronic one. Films taken after a sinus has evacuated itself or when the nose has been subjected to shrinkage by cocaine may not be expected to show an opacity suspected clinically.

Since the earliest roentgenologic evidences of pathologic changes may be first seen in the bones, films of excellent quality and fine detail are necessary to bring out changes in density. Pus, neoplasms and granulations are often hard to differentiate by roentgenograms, and clinical help must be obtained. Films of rudimentary or absent sinuses may be misleading, though lateral views and stereoscopic views perhaps with a probe in situ may be helpful.

Films indicate only alterations in normal densities, and diagnoses should be made only in terms of such densities. Fluid levels and the presence of opaque mediums should be especially noted. But lack of confidence in x-ray films is usually due to indiscriminate interpretations, poor films, failure to correlate clinical and roentgenologic findings and the eagerness to interpret pathologic entities without adequate clinical evidence. Postoperative changes may be misinterpreted. The rhinologist should be able to read the films himself in order to appreciate adequately

a report on them. Long experience and repeated observations are necessary, and teamwork between the clinician and the roentgenologist is absolutely essential.

ROENTGEN DIAGNOSIS VERSUS CLINICAL DIAGNOSIS. DR. SAMUEL R. SKILLERN, JR., Philadelphia.

The rhinologist should be capable of a correct interpretation of the roentgen report, should draw his own conclusions from it before reading the roentgenologist's report and should realize the limitations of the film as a single diagnostic factor.

The crux of the situation is the study of the film and the correlation of findings with clinical evidence. In the event that the rhinologist's conclusions differ from those of the roentgenologist, they should go over the situation in conference. In short, cooperation, consultation and coordination are the three great essentials for a successful harmony between these two diagnostic branches of medicine. It is naturally advantageous if the rhinologist can always have his films taken and interpreted by the same roentgenologist.

Many times films of the ethmoid will show clouding, even in the absence of clinical symptoms, and in the same way one may have symptomatic, hyperplastic ethmoiditis which does not show any changes in the nasal mucosa and which can be diagnosed only by roentgenograms. The same is often true of the maxillary antrum. For this reason, one must not accept an x-ray film as indicative of the need of an operation unless symptoms warrant it, since such films are as subject to error as are clinical findings.

ANATOMIC STUDIES OF EXTENSION OF A MASTOID INFECTION INTO THE DIGASTRIC MUSCLE AND ADJACENT STRUCTURES. DR. EDWARD F. ZIEGELMAN, San Francisco.

Extension outward of mastoid infection is less common than extension inward: first, because of the thickness of the external cortex; second, because a good many mastoids are of the sclerosed type, and third, because vascular drainage by the veins is inward toward the cranial cavity. External infection is usually manifested by subperiosteal abscess, zygomatic abscess or invasion of the tip muscle such as the sternomastoid. An important landmark externally is the digastric groove, and this is seen internally as the facial tip ridge or digastric ridge. This ridge is important because in extension externally by way of cells internal to this tip the digastric muscle may be involved, and if by way of cells external to this tip more often the sternomastoid is invaded. This is because there is so often sclerosis of those portions of the external tip cells which lie adjacent to the digastric ridge. Infection extending into the digastric muscle advances forward until stopped by fascial planes, evidenced by swelling of the lateral pharyngeal wall; later, it may bulge beneath the sternomastoid muscle. Infection may also occur in the muscle by invasion by continuity of tissue.

A typical Bezold's abscess may extend into either the digastric or sternomastoid muscle, while the so-called "abscess of Mouret" extends into the latter muscle. Signs are those of painful pre-auricular swelling and swelling of the submaxillary with difficulty in opening the mouth. There are the usual signs of sepsis, and the condition must be differentiated from parotitis and furunculosis. There it may be a complication following a simple mastoidectomy, and there may be seen a swelling of the floor of the mouth and lateral pharyngeal wall.

Treatment is surgical drainage by way of the submaxillary approach (Mosher).

EFFECT OF LIQUID PETROLATUM AND SOLUTIONS OF EPHEDRINE ON THE NORMAL MUCOUS MEMBRANE OF THE RABBIT. DR. WILLIAM B. STARK, Rochester, Minn.

The common use of oily solutions in the nose is the occasion for this investigation. No special harm from this is evidenced in the human being, but the practice of using irrigations of saline solution for many nasal conditions appears disadvantageous. It is well known that rhinitis frequently arises from bathing and diving. The use of irrigations after sinus operations often leads to a waterlogging of the tissues.

It was found that irrigations of sodium chloride in rabbits led to sinusitis in 75 per cent of the animals. Microscopic mucosal changes were also evident, all pointing to the undesirability of continuing this practice.

The present popularity of oily or watery solutions of ephedrine prompted experimentation with ephedrine in oil and in water in rabbits and also with liquid petrolatum.

The rabbit's nasal epithelium is much like that of man, having stratified columnar epithelium with ciliated surface cells. Below is a basement membrane of varying thickness under pathologic conditions. Glands of a mixed type with small ducts opening on to the surface lie below this layer. The various solutions were introduced into the nose with a catheter every day for a varying number of days. Later specimens of the nasal mucous membrane were taken and prepared for microscopic study. The lungs and sinuses were similarly examined.

In rabbits in which liquid petrolatum alone was used, those in which the largest injections were made showed purulent sinusitis on gross examination. The nasal mucosa of the other animals showed little change beyond slight increase in mucous secretion. Sections of the mucosa showed increase in fat according to the number of injections in the nose.

In animals that had injections of aqueous ephedrine solution, there was almost no gross pathologic process and little change microscopically. Animals that had injections of ephedrine in oil showed no gross sinusitis or pneumonia. Sections of nasal mucosa showed some increase in fibroblasts and some fat in the submucosal layer, but less than in the cases of those sections into which plain liquid petrolatum was injected.

THE THEORY OF FOCAL INFECTION: APPLICATION. DR. AUSTIN A. HAYDEN, Chicago.

An infection localized at one point in the body may produce symptoms in distant organs and tissues, though the original portal may have healed before the onset of secondary symptoms. This fundamental theory of focal infection was advanced by Billings some twenty-five years ago. The most obvious type is a multiple acute suppurative arthritis occurring during a puerperal or wound infection with a bacteremia and positive blood cultures, showing the same organism as found at the portal of entry. The second type is that of an acute osteomyelitis without any obvious portal of entry but with a history of furunculosis of ten days' duration with staphylococcus bacteremia. A third type is exemplified by typhoid fever with rose spots and Brodie's abscess of the spine with typhoid bacilli and typhoid bacteremia. All these take place through blood transportation.

A fourth type is seen in gonorrheal arthritis with a posturethral discharge. Puncture of the knee shows gonococci, and blood cultures are negative. This type of infection may be due to a short-lived bacteremia or may be of toxic origin.

A fifth type is exemplified by acute follicular tonsillitis, which, having healed, is followed by a flare-up in the joints. The tonsils must be searched as a focus of infection and the joints treated independently.

A sixth type is seen in acute rheumatic fever. There is a great variation in bacteriologic findings in the blood, some men finding many positive cultures while others do not. Both groups are probably right. Bacteremia may occur without symptoms, and their organisms may be in the blood for only a short time. One may even get positive blood cultures in normal healthy persons. Removal of the focus should benefit the distant symptoms, but may be only one factor. Others may be trauma, errors of metabolism, gout, low thyroid activity, senility and heredity. After the focus has been cleaned up, recurrence may be due to an allergic reaction in the tissues which have become sensitized to that particular infective agent.

BOOK REVIEWS

Diseases of the Nose, Throat and Ear: For Practitioners and Students. Edited by A. Logan Turner, M.D. With the Collaboration of J. S. Fraser, W. T. Gardiner, J. D. Lithgow, G. Ewart Martin and Douglas Guthrie. Third edition, revised and enlarged. Price, $6. Pp. 465, with 250 illustrations and 19 plates, of which 8 are in color. New York: William Wood & Company, 1932.

This is the third edition of this excellent textbook, which made its first appearance in 1924. Each section has been revised, and portions have been rewritten, especially the portion dealing with peroral endoscopy and the Ménière symptom complex. The outstanding addition is a description of the physiology of the otolith apparatus and its relation to clinical investigation. A number of new illustrations have been added, including skiagrams of the paranasal sinuses, the steps of the Howarth operation on the frontal sinus, the intranasal operation on the ethmoid cells, the anatomy of the pharynx, direct examination of the larynx, bronchi and esophagus, diseases of the esophagus and the pathologic changes in mastoiditis.

The statement regarding the designation of the tuning forks is still confused, as was the case in the first edition. On page 275, the fork vibrating 32 double vibrations is designated as C^2, and the one vibrating 64 double vibration as C^1; then it goes on with c, 128 double vibrations; c^1, 256 double vibrations, etc., which is correct nomenclature for these forks, leaving out entirely the C fork, which is the one vibrating 64 double vibrations. The fork vibrating 32 double vibrations is C_1, and the fork vibrating 16 double vibrations is C_2.

The reviewer is glad to see an especially lucid statement regarding the indications for operations on the mastoid and the treatment of chronic purulent otitis media. It is not clear why, under the caption of otosclerosis, the term "dry middle ear catarrh" is used as though the two terms were synonymous. In the text, otosclerosis seems to be considered as a pathologic entity. The fact that otosclerosis with changes in the body capsule of the labyrinth is occasionally found in connection with evidences of a chronic tubotympanic catarrh is hardly justification for assuming that an etiologic relationship exists between the two processes or that the two processes should be considered under the same heading. The same association between otosclerosis and suppurative otitis media is occasionally observed, but one would hardly use the term chronic suppurative otitis media as synonymous with otosclerosis. The author has not suggested that otosclerosis and chronic otitis media are synonymous terms; nor is it logical to assume that chronic suppurative otitis media is a factor in the etiology of an otosclerotic process.

Throughout the book the authors have been scrupulously careful to apply the correct anatomic terms to the various structures. The reviewer would point out a single exception in the consistent use of the term "external acoustic meatus" for meatus auditorius externus. The correct use of the term acusticus is in connection with the internal meatus, the meatus acusticus internus.

Reviews of the earlier editions of this excellent textbook appear in the ARCHIVES (**1**:129 [Jan.] 1930; **7**:196 [Feb.] 1928).

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. E. Musgrave Woodman, 22, Newhall St., Birmingham, England.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.
Place: London. Time: May 5, 1933.

Royal Society of Medicine, Section of Otology

President: Dr. J. F. O'Malley, 6, Upper Wimpole St., W. 1, London.
Secretary: Dr. C. Gill Carey, 106, Harley St., W. 1, London.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 8-10, 1933.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.
Place: Chicago. Time: June 8-10, 1933.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 9-11, 1933.

SECTIONAL

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.
Place: Indianapolis. Time: Dec. 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Walter Bristow, Medical Arts Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. G. Reaves, 422 W. Cumberland Ave., Knoxville.
Secretary: Dr. Ralph O. Rychener, 1720 Exchange Bldg., Memphis.
Place: Nashville. Time: April 10, 1933.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Tex.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Tex.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

Acting President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, September to May, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.